ANNUAL MEETING, MAY 21, 22 and 23
HOTEL STATLER, BOSTON

The New England Journal of Medicine

Formerly
The Boston Medical and Surgical Journal
Established 1828

Volume 234 APRIL 25, 1946 Number 17

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Owned and Published by the Massachusetts Medical Society

Published weekly at 8 Fenway, Boston 15, Massachusetts.
Domestic, $6 per year, 25c per copy (medical students, $3.50 per year); Canada, $7.04 (Boston funds); foreign, $8.52.
Entered as second-class matter, November 16, 1932, at the post office at Boston, Massachusetts, under the Act of March 3, 1879.
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The New England
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Volume 234 APRIL 25, 1946 Number 17

THE PROBLEM OF NUTRITION IN THE POSTOPERATIVE CARE OF ABDOMINAL WOUNDS OF WARFARE*

Lieutenant Colonel J. E. Dunphy, M.C., A.U.S., Major Stanley O. Hoerr, M.C., A.U.S., Captain C. L. Dimmler, Jr., M.C., A.U.S., and Captain Robert R. White, M.C., A.U.S.

RAPID evacuation, the improved treatment of shock and skilled surgery available within a few miles of the front have made it possible to operate on many patients with abdominal wounds of an extent and gravity seldom seen in those treated in World War I. Despite this fact, the immediate postoperative mortality of 50 per cent, generally accepted as representative of the last war,[1] has been considerably reduced.[2, 3] Most reports of experiences in this field have dealt largely with the operative and immediate postoperative periods. The complex problems in nutrition and sepsis presented by some of these patients on arrival at fixed hospitals have not been emphasized.

During the campaign in Normandy, the normal channels of evacuation for postoperative abdominal cases by-passed this unit, which received only slightly wounded men who could be promptly returned to duty or seriously wounded ones whose condition was so precarious that evacuation to the United Kingdom was impossible. In the latter group were 16 patients gravely ill with extensive abdominal wounds. These patients were selected for transfer to this unit specifically because of their poor general condition, and represent only the more complicated end results in a series of about 150 cases.

The nature and severity of the injuries are significant (Table 1). There were multiple injuries in every case. The small bowel was perforated in 9 cases, the colon in 6 cases, and the rectum in 1 case. There were liver wounds in 3 cases, a duodenal wound in 1, and a gastric wound in 1. The bladder was involved in 2 cases and the urethra in 1. In 2 cases there were combined abdominal and thoracic wounds. Ten patients had extensive soft-tissue injuries, with buttock or perineal wounds in 6 of them. There were 7 cases of compound fracture, which involved the sacrum in 4 cases and the humerus, forearm and femur in 1 case each. One patient required a high amputation of the thigh because of destruction of the external iliac artery and vein. Resection of the bowel had been necessary in 6 cases. In 1 of them, both the ileum and the colon had been resected, and in another, three separate resections of the small bowel had been performed.

*From the Surgical Service of the 5th General Hospital, United States Army.

Postoperative Complications

All 16 patients had varying degrees of anemia and hypoproteinemia when they reached this unit. There were 5 patients with external fistula, which involved the genitourinary tract in 3 cases and the duodenum and ileum in 1 each. Infection — retroperitoneal, peritoneal or in the wound — constituted a major complication in 12 cases. It was the cause of the 2 deaths in this group, in one case from rapidly progressing peritonitis and on the other from extensive retroperitoneal abscesses. Intestinal obstruction developed in 11 cases and required reoperation in 2 of them. There were 7 cases of jaundice. Azotemia was noted in 2 cases. Thrombophlebitis occurred in 2 cases, and was accompanied by a small pulmonary infarction in 1 of them. Vein ligation was not done in this case, and the episode did not recur. Three patients had residual pulmonary infection from an underlying atelectasis. Unexplained massive hemorrhage from the lower gastrointestinal tract occurred in a patient who had an extensive liver injury and moderate jaundice. No prothrombin determination could be done, and sigmoidoscopy and barium studies of the colon disclosed no abnormalities. Despite two episodes of bleeding, the patient steadily improved and was evacuated in good condition.

A discussion of the management of all these complications is not within the scope of this paper. Each was dealt with according to well established surgical principles. Maintenance of nutrition in certain patients presented considerable difficulty, however, and it is to a consideration of this problem that this report is directed.

Nutritional Status and Parenteral Fluid Therapy

At the time of admission to this unit all 16 patients were suffering from varying degrees of anemia

and acute malnutrition. Data on a representative number of cases are given in Table 2, which shows the amount of blood and plasma administered prior to arrival and the hemoglobin and serum protein levels on admission. It is evident that these patients would have benefited by a higher protein intake in the postoperative period prior to arrival.

Previous experience with abdominal wounds had convinced us of three facts — that profound anemia and nutritional edema develop with extraordinary rapidity, that large amounts of protein are required to maintain a reasonable nutritional balance and that it is much better to prevent than to treat the nutritional disturbances that develop. To meet this problem a routine nutritional program for abdominal sepsis was adopted (Table 3). This regime is essentially the same as that recommended some years ago by Coller and Maddock[4] for salt and water balance, except that it substitutes 1 liter of protein-carrying fluid for 1 liter of 5 per cent dextrose and water. It provides approximately 50 gm. of protein and 125 gm. of carbohydrate daily. If there is a considerable loss of salt, the total carbohydrate intake may be as high as 200 to 300 gm. The daily parenteral caloric intake may thus vary between 800 and 2000, depending on the amount of glucose given. The well known impracticability of repeatedly giving concentrated solutions of glucose by vein precluded its use in these cases. Thus, when the caloric intake was low it became necessary to rely on small supplemental oral feedings, usually in the form of sweetened milk. These were often begun before all evidence of peritonitis or intraperitoneal infection had subsided. Available vitamin preparations were given routinely as shown.

This regime effectively restored the levels of hemoglobin and serum protein to normal in all the patients in an average of seven to ten days. The results of therapy in Cases 3 and 7 are shown in Figures 1 and 2, respectivele. These are typical responses. The need for whole blood as well as plasma is illustrated in Case 7. In general, the more septic the patient was the slower was the response to therapy, but even in the sickest patients, whose cases are presented in detail below, the levels of hemoglobin and serum protein returned to normal within fourteen days.

The hazards of repeated large amounts of blood and plasma constitute an objection to this nutritional regime. In this series of 16 patients, fresh — one to seven days old — banked blood preserved in a sodium citrate-glucose mixture was used. Most of the blood was Type 0. No serious transfusion reactions were encountered, and there was no evidence of massive hemolysis of either the patient's or the transfused blood. There were 7 cases in

TABLE 1. *Nature and Extent of Injuries.*

Case No.	Cause of Wound	Nature of Abdominal Injury	Part Affected	Additional Injuries and Complications
1	High-explosive shell	Perforating	Colon and buttocks	Compound fracture of sacrum
2	High-explosive shell	Penetrating	Colon and ileum	Wound of right thigh
3	Machine-gun bullets	Perforating (multiple)	Liver (multiple wounds)	Compound fracture of humerus
4	High-explosive shell	Perforating	Colon and ileum	Extensive wounds of abdominal wall
5	High-explosive shell	Perforating	Buttock, perineum and urethra	Compound fracture of radius
6	Rifle bullet	Penetrating	Duodenum and ileum	Extensive retroperitoneal hemorrhage
7	Rifle bullet	Penetrating	Right pleura, diaphragm and liver	
8	Rifle bullet	Perforating	Jejunum, colon and liver	
9	High-explosive shell	Perforating (multiple)	Ileum (multiple wounds)	Extensive wound of buttock; compound fracture of femur.
10	Machine-pistol bullet	Perforating	Left pleura, diaphragm, stomach, colon, spleen and pancreas	
11	High-explosive shell	Penetrating	Colon, rectum and bladder	Wound of buttock; compound fracture of sacrum; extensive retroperitoneal hemorrhage.
12	High-explosive shell	Penetrating	Ileum, jejunum and colon	Extensive wound of buttock; compound fracture of sacrum.
13	High-explosive shell	Penetrating	Ileum (multiple wounds)	Wound of buttock; extensive retroperitoneal hemorrhage.
14	High-explosive shell	Penetrating (multiple)	Ileum, jejunum and bladder	Extensive wound of buttock; compound fracture of sacrum.
15	Rifle bullet	Penetrating	Ileum (multiple wounds) and mesocolon	
16	High-explosive shell	Penetrating	Bladder and external iliac artery and vein	Extensive soft-tissue wounds; compound fracture of pelvis.

which jaundice developed, but the available evidence indicated that severe sepsis rather than hemolysis was the principal contributory factor.[5] In 2 cases, moderate nitrogen retention without a true renal shutdown occurred. In one of these, the transfusions were continued and recovery ensued as soon as sepsis was controlled. In the other, death occurred from generalized toxemia and sepsis. Post mortem the kidneys were grossly normal. Although histologically there was minimal evidence of so-called "hemoglobinuric nephrosis," the changes were not sufficient to be regarded as a significant contributory factor to death.

Table 2. *Previous Therapy and Laboratory Data on Admission.*

Case No.	Time of Administration	Previous Blood and Plasma Therapy		Time Interval	Laboratory Data	
		Amount of Blood	Amount of Plasma		Hemoglobin	Total Protein
		cc.	*cc.*	*days*	%	*gm./100 cc.*
1	Preoperative	1000	500	16	71	5.1
	Postoperative	1000	500			
2	Preoperative	500	None	11	71	5.1
	Postoperative	4500	5000			
3	Preoperative	500	None	12	58	4.1
	Postoperative	None	500			
6	Preoperative	?	?	7	58	6.2
	Postoperative	1500	2000			
7	Preoperative	1000	1500	9	73	5.3
	Postoperative	None	None			
12	Preoperative	2000	2000	7	56	—
	Postoperative	1000	1000			
16	Preoperative	500	250	15	61	4.8
	Postoperative	1000	1000			

Although our experience with repeated transfusions of Type 0 whole blood and pooled dried plasma was uniformly satisfactory, there is increasing evidence that this therapy, especially if high-titer blood is used, produces hemolytic anemia.[6] Therefore, whenever possible, matched blood or type-specific plasma should be used. Indeed, albumin may eventually prove preferable to plasma for nutritional purposes.

In contrast to the prompt rise in hemoglobin and serum protein that occurred under this regime, the general nutritional status of the patients improved only when residual infection had been eradicated.

The Role of Infection

The principal cause of the severe degrees of malnutrition encountered in these patients was infection. In every case in which residual abscesses were easily accessible and could be promptly drained, rapid convalescence resulted. In all cases in which there were deep seated peritoneal or retroperitoneal abscesses that could not be well localized or readily drained, convalescence was prolonged and progressive wasting of the subcutaneous tissues occurred. This was true even in patients who were able to take fairly large amounts of food by mouth. The degree of weight loss in some patients was appalling; in several cases it amounted to over 60 pounds. It is therefore evident that the management of residual infection must be regarded as an essential feature of any nutritional program.

Table 3. *Guide to Daily Nutritional Requirements.**

Requirement	Form Given	Amount	Calories
Basic salt	Physiologic saline solution	500 cc.	—
Basic water (to replace loss in urine and insensible fluid loss)	5 per cent glucose in distilled water	±2500 cc.	500 (125 gm. of carbohydrate)
Replacement for salt and fluid loss from gastric suction, ileostomy, fistula and so forth	5 per cent glucose in saline solution	Variable (volume for volume replacement of lost fluids)	Variable (200 per liter)
Protein	Plasma and whole blood†	1000 cc.	200 (50 gm. of protein)
Vitamins	Vitamin B complex	50 mg.	—
	Vitamin C (ascorbic acid)	100 mg. (50 mg. twice a day intramuscularly)	—
	Liver extract	1 cc. (intramuscularly)	—
	Vitamin K‡	10 mg.	—

*This regime is not presented as the ideal one; it was the one employed on the basis of the supplies available.
†All whole blood if hemoglobin level below 80 per cent.
‡Given if jaundice or intestinal fistula present.

In dealing with infection, the following general principles were adopted. All localized, easily accessible abscesses were promptly drained. This was done even before the serum protein and hemo-

globin levels had been restored to normal. In the case of deep seated and poorly localized infection, a more conservative policy was adopted. It is well known that the peritoneum has a remarkable natural ability to deal with residual abscesses; accordingly, the patient was placed on the nutritional regime outlined in Table 2. Parenteral penicillin was given in amounts of 30,000 units every two hours, and the course of the disease was carefully observed. If the infection became well localized

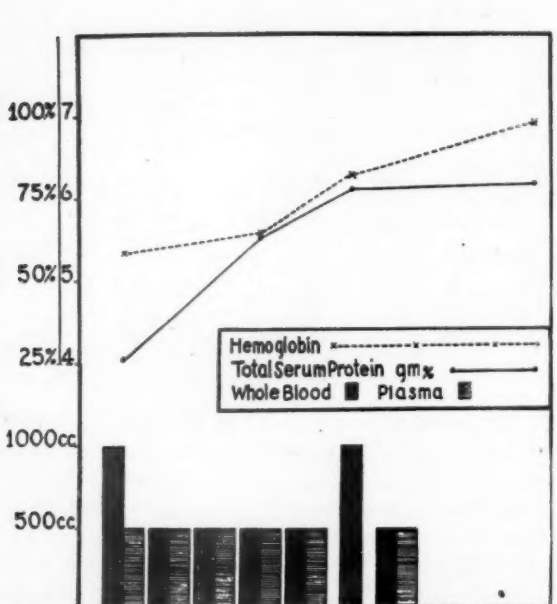

FIGURE 1.

and easily accessible through "pointing," as in a pelvic abscess, it was drained. If no localization occurred but there was a gradual and steady improvement in the patient's condition, as evidenced by a rise in the hemoglobin and serum protein levels, a decline in temperature and an improvement in appetite, a conservative policy was continued. In 3 cases, complete resolution of intraperitoneal sepsis occurred under this regime and no surgical intervention was required. If, on the other hand, no improvement occurred or if the patient appeared to be losing ground, operation was carried out regardless of the risk it entailed, provided there was reasonably accurate localization of the infection by clinical signs. The following case report is illustrative.

CASE 6. The patient received a perforating gunshot wound in the right upper quadrant of the abdomen on July 31, 1944. He was operated on 6 hours after injury and was found to have a perforation of the ileum, which was closed by suture. The bullet was felt in the retroperitoneal tissues in the region of the second portion of the duodenum. Its removal was followed by a severe hemorrhage, which was finally controlled by packing. The abdomen was closed, the pack being left in the wound.

The patient was transferred to this unit on the 9th postoperative day. He had a severe anemia and was profoundly septic, the daily temperature varying between 101 and 104°F. The packs were still in the wound, in which there was considerable deep infection, with marked digestion of the surrounding skin. There was mild jaundice. Examination of the abdomen revealed tenderness and a sense of resistance in the right upper quadrant on deep palpation that suggested a mass.

Initial therapy consisted of multiple plasma and whole-blood transfusions, in accordance with the program outlined in Table 3. Removal of the pack in the wound produced no hemorrhage, but drainage became more profuse than ever, and at times appeared to be pure bile. Despite meticulous and frequent dressings, surgical drainage of the wound and the application of suction to the fistulous tract, the wound was slowly undermined by progressive infection. Although there was a gradual rise in the hemoglobin with multiple transfusions, the patient remained profoundly septic, and his general condition was obviously deteriorating.

On August 23, 24 days after the injury and 15 days after admission, the jaundice became intense. The patient refused all food by mouth and became markedly listless and apathetic. Part of this reaction may have been due to the disturbances of acid-base metabolism caused by the high intestinal fistula. Carbon dioxide determination could not be done, but there was no tetany, the urinary output was good, and the hemoglobin and serum protein levels continued to show an upward trend. The possibility that the multiple whole-blood transfusions had produced the jaundice was raised by some observers. The patient had had severe chills and sweats with the marked swings of temperature. but none of these had been closely related to a whole-blood transfusion, and there had been no hemoglobinuria. There was bile in the urine. It was reasoned that there was an incompletely drained abscess in the right upper quadrant, that this partially accounted for the jaundice,[5] and that the duodenal fistula would not heal or subside until it was drained. Until the duodenal drainage subsided the wound infection could not be expected to clear up.

Accordingly, on August 26, under local anesthesia, the right upper quadrant was explored through an oblique subcostal incision, the original wound, which was in the midline, being carefully avoided. In the right subhepatic space there was a huge cavity filled with the same type of fluid as that draining from the wound. It was impossible to demonstrate a communication with the original wound, but the exact limit of the cavity could not be felt. All fluid was removed, and the cavity was drained.

Following this operation there was steady improvement. The patient became more alert and resumed eating, and the jaundice began to diminish. It is interesting, however, that the temperature went higher than before, ranging between 101 and 105°F. Since the patient's general condition appeared improved the febrile reaction was disregarded, and at the end of 2 weeks the temperature began to decline. The fistula was diverted from the original badly infected abdominal wound to the oblique subcostal incision, and the original wound rapidly cleared up and healed. By vigorous efforts on the part of the nursing staff, the second wound was kept clean and in good condition despite the excessive amount of drainage, and it gradually healed, leaving only a small fistulous tract. Six weeks after operation the patient was ambulatory and afebrile. The wounds were healed, but there was still a small fistula, which on lipiodol injection was found to enter the second portion of the duodenum. It seemed likely that this would heal without further intervention.

The patient was evacuated in excellent condition. He made steady progress and was ready for return to duty 8 months after injury. The fistula healed spontaneously.

Patients with undrained areas of infection that cannot be readily localized present a still more baffling problem. There were 3 patients in this category, 2 of whom died. One death was due to rapidly progressive peritonitis. The patient had received extensive shell wounds of the left side of

the chest, spleen, stomach, colon and pancreas. Post-mortem examination disclosed gross leakage from all the suture lines in the multiple wounds of the stomach and colon. The other death occurred as a consequence of long-standing intestinal obstruction associated with a large undrained retroperitoneal abscess. Although the intestinal obstruction was relieved by surgery after admission to this hospital, the patient gradually lost ground and

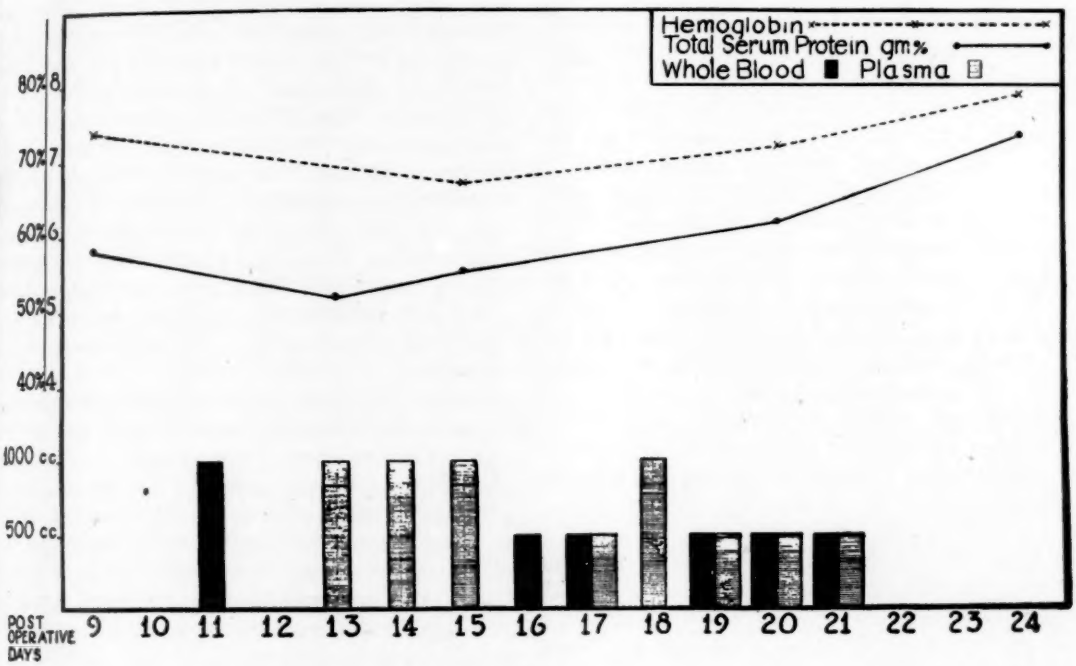

FIGURE 2.

died of sepsis eight days later. The third case in this group is illustrative of this type of problem and is of sufficient interest to merit a detailed presentation.

CASE 2. The patient was wounded on July 25, 1944, when a high-explosive shell fragment entered the anterior abdominal wall just to the right of the midline below the umbilicus. He was operated on 6 hours after injury at a field hospital under Pentothal Sodium anesthesia, and was found to have perforating wounds of the ileum and colon. A resection of the ileum, with end-to-end anastomosis, was done. The wound in the sigmoid was repaired with a purse-string suture, and a Mikulicz colostomy was performed proximal to the area of injury. A partial traumatic amputation of the left 5th finger was completed, and a wound of the right thigh was debrided.

The patient did well up to the 11th postoperative day, at which time he was given prostigmine. This was followed by signs of acute intestinal obstruction. On the following day, the abdomen was reopened and obstruction at the site of the anastomosis was found. A double-barreled enterostomy was done at the point of obstruction. Postoperatively the patient did poorly, although there was evidence of relief of the obstruction. He was transferred to this hospital on the fourth day after the second operation.

Physical examination on admission showed an acutely ill, extremely emaciated patient. There was slight jaundice and moderate peripheral edema. There was a double-barreled enterostomy through a right rectus incision, surrounded by gross infection. The midabdominal incision through which the original operation had been performed was grossly infected. Peritoneal or retroperitoneal infection was manifested by tenderness in the right lower quadrant and the lower abdomen. No definite abscess was apparent. The long wound in the right thigh involving the adductor muscles was moderately infected. There was a large sacral decubitus. The colostomy wound was clean, but the colostomy was not functioning.

Laboratory studies showed the serum protein to be 5 gm. per 100 cc. and the hemoglobin 61 per cent, despite moderate dehydration. The patient had received sulfadiazine parenterally up to the time of admission.

Treatment consisted of daily infusions of blood or plasma, penicillin parenterally and frequent dressings to control infection and prevent digestion of the skin around the ileostomy. Although there was an ill-defined inflammatory mass in the right lower quadrant, the presence of the ileostomy, the profound nutritional disturbances and the patient's poor general condition rendered exploratory search too hazardous. The patient seemed improved one day and worse the next, and became progressively more jaundiced.

Despite supportive measures, the patient gradually lost ground, becoming irrational and semicomatose by the 15th hospital day. He showed evidence of nitrogen retention, the highest nonprotein nitrogen level being 84 mg. per 100 cc., despite a parenteral fluid intake of 3000 to 4000 cc. daily and a urinary output of 1500 to 2000 cc. The wound in the right thigh seemed to be developing a deep cellulitis. It was thought that this was due to retroperitoneal infection extending downward along the fascial planes and that the wound in the thigh might have continuity with the abdominal incision, but although the purulent discharge of the abdominal and thigh wounds was similar, actual continuity was never proved. The increase in drainage from the thigh coincided with an abrupt end of the downward course, and from this point on gradual improvement was noted. The icterus slowly cleared. Eventually further surgical drainage of the thigh was carried out. The spur on the double-barreled colostomy was cut down, and the wound infection cleared.

The patient soon began to take food by mouth, and from this time on his progress was steady. The jaundice cleared entirely, the azotemia subsided, and the blood picture returned nearly to normal. On October 4, an attempt was made to cover the large sacral ulcer with skin. Although the granulations appeared healthy, a split-thickness graft failed to take. On October 18, an attempt was made to close the ileostomy under local anesthesia. No attempt was made to free the bowel because it seemed that the patient's general condition would not withstand an extensive procedure, but the closure broke down 36 hours after operation. Despite the leaking ileostomy he continued to improve, and he was evacuated in fair condition. Subsequent reports indicate that he made a slow but steady recovery.

The management of this type of case remains a problem, but it is our belief that if improvement does not occur under so-called "conservative" management, an operation to find and drain the infection must be undertaken even if it requires an exploratory laparotomy. In this case a more aggressive policy, with drainage of the peritoneal infection and closure of the ileostomy at an early date, might have been wiser. If spontaneous drainage of the retroperitoneal infection had not occurred, the patient would probably not have recovered. In this type of case, in which convalescence is prolonged by deep-seated, poorly localized infection, the maintenance of nutrition is exceedingly difficult. It is comparatively easy to prevent hypoproteinemia and anemia by frequent transfusions of blood and plasma, but despite this fact there is progressive weight loss and wasting of the subcutaneous tissues. Small amounts of highly nutritious liquids may be given by mouth, but at times these are not well tolerated, and even when they are it is difficult to raise the caloric intake above 2500 daily. Under such circumstances the use of protein digests, which were not available, would be desirable.[7, 8]

External Fistulas

There were 5 patients with external fistulas; — in the urinary tract in 3 cases, in the duodenum in 1, and in the ileum in 1. The urinary fistulas were relatively innocuous so long as there was adequate external drainage. The 2 patients with intestinal fistulas, whose cases have been presented in detail, presented extremely serious problems. It is significant, however, that once infection was controlled their general condition improved despite the presence of the fistula, and in 1 case the fistula healed spontaneously. The difficulties encountered in the case in which an ileostomy had been performed support the contention of the surgeons who maintain that ileostomies cause more deaths than cures. Also, it is evident that if a patient is losing ground, a surgical attack on the fistula should be undertaken early, before his condition has deteriorated to such a point that even a limited procedure cannot be tolerated.

Chemotherapy

All these patients had received sulfanilamide in the wound and sulfadiazine and penicillin parenterally prior to admission to this hospital. Ther was considerable variance in the dosages used and so far as could be determined from the record some irregularities in the schedule of administra tion. In general, however, all the patients ha received combined therapy in reasonably adequat dosages over periods of seven to sixteen days. O arrival at this hospital the sulfonamides were di continued. This was done for several reasons. Firs it was argued that whatever residual infection the patients showed had developed despite the use o the drugs, and that therefore their continued us was not likely to be beneficial. Second, the probabl nature of the infection — namely, a localized co lection of undrained pus — made it unlikely tha sulfonamide therapy would affect it. Third, th continued use of sulfonamide drugs, with the tendency to produce toxic and febrile reaction would add an additional factor to cases already c complex that interpretation of the cause of feve was difficult. Finally, and most important, it seeme unwise to continue the use of these drugs in patient with such profound metabolic disturbances — tha is, dehydration, anemia, hypoproteinemia, azotemi and jaundice.

The sulfonamide drugs were discontinued becaus of certain specific objections to their use. Since n such objections were known to pertain to the use o penicillin, and since it seemed unwise to discontinu all chemotherapy, this agent was employed routinel in large amounts — 30,000 units every two hours We were influenced in this decision by two things First, the controlled observations of Lyons[9] in dicate that the nutritional status of patients wit septic wounds is restored to normal more rapidl when penicillin is given, as well as blood and plasma Second, our previous experiences with the use o this drug had revealed dramatic improvement afte its use in several patients gravely ill with long standing abdominal sepsis.

Since penicillin was used routinely without con trol periods of observation, there are no data o which to base an appraisal of its value. Although it was given in large amounts over a period of many days and weeks, — one patient received 4,000,000 units, — no toxic reaction was observed. The re peated hypodermic injections became an ordeal for some patients, particularly those whose con valescence was prolonged. It is our impression, based only on a comparison between the course of these cases and our recollection of the course of similar cases treated without penicillin, that the drug was of value.

Results

The nutritional state of 9 of the 16 patients rap idly improved under the regime outlined in Table 2 These were the patients in whom sepsis wa promptly eradicated by adequate drainage. Three patients in whom residual peritoneal infection sub

sided under conservative therapy also made steady, although less dramatic, improvement. In these 12 cases the nutritional regime employed can be described as adequate. In 2 of the remaining 4 cases, convalescence was greatly prolonged by residual, poorly localized infection and by the presence of external intestinal fistulas. Under these circumstances, although anemia and hypoproteinemia were prevented, the patients steadily lost weight on the nutritional regime employed. As soon as infection was eradicated, however, they promptly improved.

Two patients died, one of overwhelming peritonitis shortly after admission and the other of inadequately drained retroperitoneal abscesses, which were not detected until the post-mortem examination. When it is considered that these cases were the most serious ones evacuated at the time from three different field hospitals, the end results may be described as gratifying.

Discussion

In the postoperative care of the patient with war wounds of the abdomen, two cardinal principles should guide the surgeon — maintenance of nutrition and eradication of infection. These are complementary, since the well nourished patient can better resist infection and, conversely, nutrition improves as soon as infection subsides.

In actual practice, the maintenance of nutrition in these patients is difficult. Despite the liberal use of blood and plasma in forward areas, the enormous losses that may accompany extensive abdominal wounds are rarely completely replaced, and the nature of the injury in itself temporarily prohibits the taking of food by mouth. Thus, unless specific corrective measures are taken every patient develops a certain degree of anemia and malnutrition. Fortunately, if a good salt-and-water balance is maintained this period of starvation is readily withstood by the majority of patients, but if complications develop, particularly peritoneal or retroperitoneal infection, external fistulas and intestinal obstruction, the patient faces these additional burdens already seriously handicapped by malnutrition. Once a complication develops, the nutritional state deteriorates at an appalling rate.

It therefore becomes essential to attempt to maintain nutrition from the beginning by some such regime as that outlined in Table 3. The additional blood and plasma are little enough, and by no means satisfy the basic caloric requirement. A perfect parenteral food substitute has not been developed, but whatever substance is available should be employed to the fullest extent. The liberal use of blood and plasma in the early postoperative period is a step in the right direction. The administration of amino acids, — hydrolyzed casein, — either parenterally or in small amounts orally is desirable in the care of certain of these patients. Nutritional regimes similar to those outlined by Co Tui et al.[7] and Elman[8] more closely meet the nutritional demands than does the regime employed in the present study. To the surgeon of the future it will seem as unwise to fail to feed these patients parenterally from the very moment of operation as it now seems to us to fail to transfuse a patient who has had a massive hemorrhage merely because he is likely to survive without transfusion.

The danger of residual infection demands constant vigilance. Adequate drainage of wounds and of local and easily accessible abscesses must be carried out promptly. In the case of deep-seated peritoneal and retroperitoneal infection, the condition of the patient must be weighed in the balance. If he is obviously getting well, the problem may solve itself. The natural ability of the peritoneum to combat residual infection is well known and is confirmed by this study. It is in the patient who is not getting well that an aggressive attitude toward residual infection must be adopted. Subphrenic and pelvic abscesses may be diagnosed with relative ease, and their management is standardized. Foreign bodies should be regarded as a possible clue to the location of obscure infection. If these sources are excluded, it is our conviction that in the failing patient an exploratory search for hidden abscesses is justified. This may indeed appear a drastic measure, and sound judgment must be exercised in appraising the given patient, but by this means certain lives can be saved that would otherwise be lost.

Since the presence of external fistulas adds a serious burden to the patient convalescing from abdominal wounds, open ileostomies should be avoided. Associated infection, however, appears to have a more deleterious effect on the nutrition of a patient than does external fistula alone. If infection can be controlled, a fistula may heal spontaneously. If a patient is losing ground, surgical repair of a fistula should not be delayed.

Summary

A report is presented of the nutritional problems encountered in a series of 16 patients with extensive abdominal wounds. The series was a selected one in that these patients were too ill to be evacuated directly to the United Kingdom during the early weeks of the campaign in Normandy.

The need for an adequate nutritional regime in the early postoperative care of these patients is emphasized. The liberal use of blood and plasma, not only before but for some time after operation, is necessary.

A routine nutritional program is presented. In cases in which residual infection was readily eradicated this program proved adequate. When there was persistent infection, convalescence was prolonged and a parenteral food substitute to increase the total caloric intake would have been desirable.

The management of residual or latent infection is an essential feature of any nutritional program. Adequate drainage of wounds and of localized abscesses must be promptly performed. A persistent search for undrained foci of infection must be carried out, and on occasion in the failing patient an exploratory operation is justified.

Since external fistulas add a serious burden to the patient with abdominal wounds, open ileostomies should be avoided. Associated infection, however, appears to have a more deleterious effect on the nutrition of a patient than does an external fistula alone. If infection can be controlled, a fistula may heal spontaneously.

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COARCTATION OF THE AORTA*

A Report of Two Cases

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SINCE the first case of coarctation of the aorta was recorded in 1791, many excellent papers concerning the nature of this interesting condition have been published. Both the infantile and adult forms, with their pathogenesis, anatomy, associated diseases, such as cerebral and other types of aneurysms, symptomatology and diagnostic and constitutional characteristics have already been ably and exhaustively reviewed.[1-5] Indeed, Lewis[4] states, "So much has been written about coarctation of the aorta that new records of cases can have little value unless they reveal new features of interest or importance to the study of the condition." This statement might well dissuade one from writing another paper on the subject, were it not for the need of calling attention to some of the uncritical diagnostic procedures that have won general usage. For example, many physicians, with a false sense of assurance, rule out the presence of coarctation of the aorta by the finding of a normal femoral pulse. A review of the literature and study of the 2 cases presented in this paper indicate that diminution or absence of the femoral pulsation in aortic coarctation is one of the less constant signs.

According to Pardee,[6] the major criteria in this anomaly are four in number. They are as follows: erosions of the margins of the ribs, absence of the aortic knob and dilatation of the ascending aorta; a high blood pressure limited to the upper extremities; a loud systolic murmur audible over the manubrium sterni and in the left interscapular area; and pulsations of the subscapular, intercostal and internal mammary arteries.

Even a cursory review of the literature reveals that almost any of these features may be absent or obscure in a given case. Thus, absence of rib crenations, especially in younger patients, is not unusual. Lack of prominence rather than absence of the aortic knob in the radiographic silhouette is frequently seen in coarctation of the aorta as well as in other types of cardiac disease. In left-oblique films, the retroaortic and retrocardiac spaces are said to be clear in cases of coarctation,[7] instead of presenting the normal haziness in this area ordinarily produced by the ascending arch and the upper portions of the descending thoracic aorta. These findings may, however, be obscured, since young persons may normally show minimal density of the aortic shadow.

Left-ventricular hypertrophy is not a regular finding, and its presence in so many other cardiovascular diseases limits its value as a pathognomonic sign in this one.

Dilatation of the ascending aorta seems to be the most constant radiologic feature, excluding, of course, angiocardiographic studies, which at present are not in widespread use. Syphilitic aortitis may produce a similar picture, but the differentiation should not prove difficult. Thus, it is seen that in ordinary radiographic or fluoroscopic examination the combination of dilatation of the ascending aorta and lack of prominence of the aortic knob is the most frequently demonstrable finding.

A high blood pressure limited to the upper extremities is one of the most constant features of the disease. The reason for the hypertension has not been unequivocably demonstrated. The theory most favored postulates an increased peripheral resistance by the coarctation and the collateral circulation as the main cause.[8] Page,[9] however, has been unable to demonstrate a consistent rise in the

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blood pressure of the upper extremities of dogs in which experimental coarctation was induced by means of Goldblatt clamps applied to the aorta. He attributes this phenomenon to the prompt and ready development of collateral channels in these animals.

King[10] has pointed out that with embarrassment of circulatory compensation the blood pressure may drop to normal levels, and he has in addition reported a case in which a normal tension in the brachial arteries was found in an asymptomatic woman, in whom he postulated the existence of an aortic coarctation. The patient presented physical evidences of collateral circulation and a blood pressure lower in the popliteal arteries than in the brachials, with dilatation of the ascending aorta the only striking radiologic feature.

Failure to palpate both radial pulsations simultaneously or to determine blood pressure as a routine in the right arm, when only unilateral readings are taken, obscures the presence of a hypertension or pulse asymmetry in cases in which the pathologic process extends to or above the origin of the left subclavian artery.[11,12] That the right arm rather than the left should be chosen for routine unilateral blood pressure determinations is a point that has been insufficiently stressed. King[10] has emphasized the work of Korms and Guinard and of Stieglitz and Propst, who have pointed out that in most persons the blood pressure in the right arm is usually higher than that in the left. The reasons given for this difference have been many, but the difference in the anatomy of the blood supply to the right and the left upper extremity is probably the most important single factor.

As pointed out by Gitlow and Sommer,[13] in cases in which the collateral circulation is well established and constant, no difference in pulse volume and blood pressure between the upper and lower extremities may be detectable by ordinary clinical means. One of the patients of Stewart and Bailey[14] showed no reduction in the blood pressure of the legs as compared with that of the arms.

The third diagnostic criterion — namely, the presence of a loud systolic murmur over the manubrium and in the left interscapular space — is of importance only in indicating the need for an extension of the search for its etiology. Nothing is pathognomonic about the murmur, since its character varies in different cases.

Pulsation of the subscapular, intercostal and internal mammary arteries, interpreted as evidence of marked development of collateral circulation, is considered one of the most valuable clues to the diagnosis of coarctation of the aorta. Although such conditions as aneurysm, intrathoracic neoplasm and certain types of exophthalmic goiter may, as pointed out by Blackford,[3] produce a similar finding the possibility of confusion is not great. Indeed, King[10] believes that clinical evidence of collateral arterial circulation is the most important single clue to the diagnosis, this sign having been present in all the 16 cases of coarctation that he has examined. In Blackford's series, collateral circulation was clinically detectable in 35 cases, was not mentioned in 15 cases, although it was implied in about half of these, and was absent in 5 cases. In one of the cases reviewed, the patient, a pilot, who while living presented no clinical evidence of superficial collateral circulation, was revealed at post mortem to have had dilated internal mammary vessels serving as the principal site of the necessary shunt.

It is apparent that the clinical detection of evidences of collateral circulation depends in great degree on which of two main routes of blood flow is utilized.[13] If there is anastomosis of the branches of the subclavian artery, such as the superior intercostal, postscapular, interscapular and subscapular arteries, together with the aortic branches of the internal mammary arteries, with the first four intercostal arteries, most of the blood thus being carried into the descending aorta and thence to the lower extremities, the evidences of collateral circulation may well be marked. If, however, a more circuitous but less superficial anastomosis — that of the internal mammary arteries with the superior and inferior epigastric arteries — occurs, clinical evidence of collateral circulation may not be so obvious. It is in the latter instance especially that a useful diagnostic procedure — namely, simultaneous palpation of the right radial and femoral pulses — is a valuable adjunct, in that a distinct lag in the summit of the femoral pulse may be felt. As a corollary, it can be stated that a diminished or absent femoral pulse[15] was noted in many of the cases reported.

The latter finding unfortunately has assumed undue prominence in the diagnostic armamentarium of many clinicians, and it is with this phase of the subject that the present paper is primarily concerned. It has been our experience that many physicians in examining cases in which coarctation of the aorta must be considered in the differential diagnosis have ruled out its presence on finding normal femoral pulsations, or pulsations in the more peripheral arteries of the lower extremities. This misconception has crept into the literature. In Musser's[16] textbook it is stated, "The blood pressure reading from the arms is usually elevated, whereas the pulsation in the femoral arteries is greatly reduced or may even be imperceptible." In a recent article, Baker[17] states that a cardinal sign of coarctation of the aorta is absence of pulsations in the peripheral arteries of the lower extremities. "If a feeble pulsation is detected over the femorals," he continues, "it is asynchronous with the radial pulse. If the pulsations of the dorsalis pedis and posterior tibial arteries are present, coarctation can be excluded as a possibility without further ado." In a paper by Green[18] it is stated to be the practice to palpate routinely for pulsations in the femoral arteries. If

these are absent, the blood pressures are taken. Others[19, 20] have also recommended palpation for femoral pulses as a necessary procedure. This practice is in conflict not only with our experience but also with that of others. Thus, Stewart and Bailey[14] discovered in their series of cases that the absence of pulsations in the arteries of the legs was an inconstant finding, not only in the femoral vessels but in the popliteal and dorsalis pedis as well.

Certainly, a sign more constant than pulse changes in the lower extremities should be relied on in routinely ruling out coarctation of the aorta. Many other writers have called attention to the basic inadequacies of femoral palpation as an absolute diagnostic criterion. Abbott[1] in her encyclopedic review points out that retardation and diminution or even absence of the femoral pulse is not constant, either in its presence or in the extent of diminution. Blumgart, Lawrence and Ernstene[8] have made a similar statement. One of their patients showed pulse waves in the femoral arteries approximately equal to those in the radials, and the radial pulse wave appeared approximately 0.05 seconds before the femoral. Differences of this small degree are not detectable by the usual gross clinical methods of palpation. Furthermore, in the case cited the popliteal and dorsalis pedis pulsations were perceptible but weak, although the posterior tibial pulses were not felt. Considering the facts that the patient was sixty-six years old and that auricular fibrillation was present as well, it becomes evident that overattention to the pulse in the lower extremities without due regard to the other more significant signs may cause one to miss the diagnosis. In pulse tracings taken by Railsbach and Dock[21] in a case of coarctation of the aorta, the femoral arterial pulse followed the radial by 0.02 seconds, a time lag certainly within the limits of subjective error of the observer.

In short, we agree with such writers as Goodson[22] that a retarded, diminished or absent pulse in the lower extremities in the presence of a full, bounding pulse in the upper extremities is pathognomonic of coarctation of the aorta provided embolism in the iliac arteries is ruled out. But this statement is accepted in the sense that this finding is pathognomonic only when present, and that coarctation not infrequently exists in its absence.

There follow the reports of 2 cases in which the femoral pulsations were easily palpable and of normal volume, with nothing in the nature of the pulsations that was considered as diagnostically significant.

Case Reports

Case 1. The patient, a 25-year-old German prisoner of war, entered a station hospital on June 28, 1944, complaining of sore throat and pain in the left side of the back over the lower ribs of 10 days' duration. He was a well developed and well nourished, broad-shouldered and narrow-waisted man of athletic build. There had been no unusual childhood diseases. The patient had experienced some dyspnea on moderately severe exertion for the last 3 years, especially when swimming. For the last few years he had noted pains in the left side whenever he engaged in unusually strenuous work or exercise. The pains were stabbing, were aggravated by movement and by bending and usually disappeared spontaneously after 5 or 10 minutes' rest. On January 30, 1944, this patient's left foot and leg were injured by the explosion of a hand grenade. He was operated on 10 hours after injury for repair of the posterior tibial artery (?) of the left leg and also for arterial repair in the medial aspect of the left thigh above the knee. Because of recurrence of bleeding a second operation was performed 10 days later.

Early in the following June, the patient was sent to pick onions on a farm, and shortly thereafter the pain in the back became more constant, with fewer intervals of freedom from pain. It was especially aggravated by bending forward. It was localized over the left lower ribs and was neither severe nor completely disabling.

Physical examination revealed traumatic and operative scars at the medial aspect of the lower third of the left thigh and the lower third of the left leg over the posterolateral area. The pharynx was slightly injected. Forceful carotid pulsations were noted in the neck, and a systolic bruit was present. Systolic bruits were heard best over both infraclavicular fossas and at the level of the 3rd rib posteriorly in the interscapular area. There was a visible pulsatile and palpable artery, somewhat tortuous in its course, over the medial and upper border of the right scapula. An occasional sibilant rale was heard posteriorly. Vascular examination revealed a posterior tibial artery pulse barely palpable on the right and nonpalpable on the left, and a dorsalis pedis pulse weakly palpable on the right and absent on the left. The femoral pulses were easily palpable and of normal volume bilaterally. The radial vessels were somewhat thickened in relation to the patient's chronologic age. The blood pressure was 180/100 in both arms and 120/100 in both legs.

Cardiac examination revealed visible arterial pulsations over the left anterior axillary area. There was accentuation of the aortic second sound, with palpable closure of the aortic valve and a blowing systolic aortic murmur. A musical systolic murmur, moderate in duration, was heard at the apical region and was transmitted to the left anterior axillary region, and also to the right toward the xiphoid process and along the left sternal border. The rhythm was regular, and there was no displacement of the apex or increased manubrial dullness. The spleen was palpable, firm and nontender and was felt 1 fingerbreadth below the left costal margin. Pulsations of the abdominal aorta were not felt.

The red-cell count was 4,370,000, and the hemoglobin 13.5 gm. The white-cell count was 5400, with 55 per cent neutrophils, 33 per cent lymphocytes, 9 per cent monocytes, 2 per cent eosinophils and 1 per cent basophils. Urinalysis, a Kahn test and a blood smear for malarial organisms were negative. The sedimentation rate was 11 mm. in 60 minutes. The heterophil antibody test revealed agglutination in a dilution of 1:112.

X-ray examination of the chest in different projections revealed widening of the base of the heart and the ascending portion of the aortic shadow. Fluoroscopic examination showed a strong impulse on the right side of the cardiac shadow and absence of a true aortic arch. Irregular crenation defects were present along the inferior margins of many of the ribs posteriorly. The bronchovascular markings were prominent, but the pulmonary structures were essentially normal. The findings described were compatible with a coarctation of the aorta. The electrocardiogram revealed a normal axis with small Q waves in Leads 1 and 2, a large Q wave in CF_4 and a diphasic T_1.

Case 2. The patient, a well developed, 22-year-old medical student, had known of the existence of hypertension for some time. Previous examinations had always revealed an initial elevated tension, which was brought down to almost normal limits by rest. The past history was noncontributory.

The blood pressure was 174/100 in the right arm and 180/100 in the left in the supine position. Femoral pulsations were readily palpable and of normal amplitude. The blood pressure in the right leg was 120/95. In the supine position there was a blowing systolic bruit, heard best over the xiphoid and transmitted to the left. There was also a blowing systolic pulmonic murmur of moderate duration and intensity. In the erect position there was heard an impure first apical sound.

Posteriorly systolic bruits were heard over the left chest. No visible palpable arterial pulsations were noted posteriorly.

Fluoroscopic examination in the posteroanterior position revealed elongation and rounding of the left lower ventricular contour, with an undulating type of pulsation at the waist of the heart. The ascending aorta was broadened, pulsations were moderately exaggerated, and the aortic knob was not so prominent as it normally is. In the right anterior oblique position, there was moderate encroachment on the retrosternal space. The width of the aorta at its uppermost margin seemed normal. In the left anterior oblique position, there was a markedly expansile type of pulsation in the region of the inflow tract of the left ventricle. The contour of this portion of the left ventricle seemed more convex than that normally seen. The ascending aorta appeared broader and more anterior in position than normal. The aorta could not be traced under the arch. X-ray examination of the chest revealed areas of crenation along the lower borders of several of the ribs. The diagnosis was coarctation of the aorta, with an enlarged heart.

The electrocardiogram revealed a rate of 78, with a sinus rhythm. A small Q_1 and inverted T_3 were present. There was a tendency to left-axis shift.

The patient in Case 1, aside from presenting what was suggestive of a subsiding infectious mononucleosis, was admitted with a dispensary diagnosis of pleurisy. Analysis of his symptoms showed that the chest pain was undoubtedly related to coarctation of the aorta. The pain was in all probability due to root or nerve pressure caused by passage of the enlarged anastomotic arteries through the intervertebral foramens.[5, 23] The patient sustained an injury of the left foot and leg from a hand grenade, and ten hours later operation was performed for arterial repair in the left thigh and repair of an artery above and posterior to the ankle. According to the patient, recurrence of bleeding at the site of the arterial repair in the thigh ten days later necessitated reoperation. This injury may explain the absence of the dorsalis pedis and posterior tibial pulsations in the left foot contrasted with the right.

Case 2 is that of an asymptomatic medical student who had previously been repeatedly examined during participation in the Army Specialized Training Program. Hypertension, which was brought down to the upper limits of normally accepted standards by rest, was known to be present. Apparently the absence of visible and palpable evidences of collateral circulation, as well as the presence of a normal femoral pulse, had previously diverted the minds of the examiners from the possibility of aortic coarctation.

The autopsy incidence of coarctation of the aorta is said to be 1:1550.[3] To increase the accuracy of an intra-vitam diagnosis, primary stress must be placed on the most constant diagnostic features, as a preliminary screening measure in ruling out the presence of aortic coarctation. That there is no one clinically detectable and universally constant feature that is easily demonstrable in the course of a routine examination has been shown. Even the almost universal finding of a blood pressure higher in the upper extremities than in the lower was absent in one of the cases reported by Stewart and Bailey.[14] Except, however, for angiocardiographic studies such as those made by Grishman et al.,[12] which do not lend themselves to widespread usage, this decrement in arterial pressure in the legs as compared with that in the upper extremities seems to us, as well as to Stewart and Bailey,[14] the most frequent and readily detectable distinctive feature of coarctation of the aorta. The determination of the blood pressure in the lower extremities, although possibly irksome to the busy practitioner, must be made in every case in which otherwise unexplained factors exist, such as bizarre types of murmurs or bruits heard anywhere in the chest, arterial hypertension or prominent or unusual arterial pulsations, regardless of the age of the patient.

At present, the value of prompt diagnosis, aside from the desire for scientific accuracy, lies in the advantage of guiding patients intelligently in forming patterns of activity and hygiene within the limits of their vasculature. Possibly, the future holds promise for more active surgical therapy.[24]

Summary

The major criteria for the diagnosis of coarctation of the aorta are reviewed and evaluated, and 2 cases are described.

A brachial blood pressure higher than the popliteal blood pressure is one of the most constant and easily detectable clinical features of the disease.

Delayed, diminished or absent femoral pulsation may be noted in many cases, but this sign cannot be relied on routinely to rule out coarctation of the aorta.

Determination of the blood pressure in the lower extremities must be made in every case in which otherwise unexplained factors exist, regardless of the age of the patient.

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PSYCHOGENIC FACTORS IN THE ETIOLOGY OF DIABETES

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EVER since the demonstration by Von Mering and Minkowski that extirpation of the pancreas in dogs produces diabetes, a great deal of research work has been performed to elucidate the pathogenesis of this disease. The influence of heredity has been stressed, and the evidence that diabetes is inherited as a Mendelian recessive has been presented by Pincus and White.[1] Additional factors, such as obesity, have also been evaluated. From the experimental field, the relation of diabetes to the pituitary gland has been demonstrated by Houssay[2] and that to the adrenal glands by Long and Lukens.[3] Recently, the study of experimentally induced alloxan diabetes[4] has opened new possibilities for research. Fundamentally, however, the factors that operate to precipitate diabetes are not clear, nor is the mechanism whereby these factors operate.

Transient glycosurias following emotional disturbances have been described. Boehm and Hoffmann[5] produced glycosuria in cats following the arousal of rage and the application of painful stimuli. Cannon, Shohl and Wright[6] reproduced these experiments without the use of painful stimuli. Folin, Denis and Smillie[7] reported that 18 per cent of 34 second-year medical students and 17 per cent of 36 female college students showed traces of sugar in the urine immediately following an examination. Glycosuria produced in this manner has been called "emotional glycosuria." It has not been shown to become permanent, nor has diabetes ever been produced experimentally in this manner.

A review of the literature concerning the possibility of producing diabetes by psychic trauma reveals some interesting differences of opinion. Clinicians with great experience in treating diabetics, such as Joslin,[8] Root[9] and Von Noorden,[10] are not convinced that such a relation of cause and effect exists. Joslin cites Von Noorden's experience in World War I to the effect that neurogenic diabetes does not occur. As a consultant for a hospital center in France through which 40,000 soldiers passed, Joslin discovered only 2 cases of diabetes, despite his known interest in this particular disease. Matz[11] studied diabetes in veterans of World War I, and came to the conclusion that neurogenic factors are of no importance in the etiology of diabetes.

On the other hand, investigators in the psychiatric field, particularly Menninger[12] and Daniels,[13] are not so convinced that the subject is closed. They believe not only that there is a correlation between mental stress and strain and increases in the severity of the diabetic state but that diabetes may be initiated by psychologic or emotional disturbances. Our interest in this phase of the problem arose after observing several patients in whom a psychogenic trauma seemed to precede the development of diabetes in such a way as to suggest cause and effect.

The records of all patients who were treated for diabetes on the Medical Service of the Lovell General Hospital between August 1, 1944, and June 30, 1945, — a total of 44 cases in 21,993 admissions, — were reviewed. All these patients were seen personally by us.

The diagnosis of diabetes mellitus in these patients was based on either one of two criteria. The first of these was the occurrence of an illness characterized by hyperglycemia — with a blood-sugar level of 300 mg. per 100 cc. or more — associated with glycosuria and occasionally with acetonuria, and necessitating a diet and moderate to large doses of insulin — 30 or more units daily — for complete control. Some of the patients had had glucose-tolerance tests done overseas and typical diabetic curves had been obtained. In others, no such test had been made, but when an attempt was made to reduce the dosage of insulin, glycosuria and hyperglycemia appeared, although the patient was on a strict diet and was taking 30 or more units of insulin a day. It was not considered either necessary or desirable to perform a glucose-tolerance test in these cases. The second criterion was as follows. In mild cases of glycosuria, glucose-tolerance tests were done, and in many cases these were repeated after the patients had been on a high-carbohydrate diet for about a week, especially if there was any doubt about the original diet. The diagnosis of diabetes mellitus was made if the blood-sugar value in the test exceeded 180 mg. per 100 cc. at its peak and did not return to normal within two hours. In

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TABLE 1. *Data in Patients with Known Precursors to Diabetes.*

Case No.	Age	Relatives with Diabetes	Height	Normal Weight	Date of Onset	Date of Hospitalization	Regime on Discharge	History of Onset
	yr.			lb.				
1	22	0	5 ft., 8 in.	180	Apr., 1944	Oct., 1944	C200-P80-F80; PZI 15 units, RI 10 units.	Gradual onset in England of polyuria, polydipsia and polyphagia, with 40 pounds weight loss
2	25	Brother	5 ft., 5 in.	172	Nov., 1944	Dec., 1944	Regular diet	Gradual onset of polyuria while overseas; patient developed laryngitis, and routine urine examination revealed sugar.
3	33	Mother	5 ft., 5 in.	195	?	Oct., 1944	Regular diet	Routine urine examination revealed sugar during hospitalization overseas for upper respiratory infection
4	27	0	6 ft.	192	Aug., 1944	Aug., 1944	C250-P80-F80; PZI 10 units.	Gradual onset of polyuria and polydipsia while overseas
5	29	0	5 ft., 7½ in.	185	June, 1944	?	C160-P100-F80; PZI 10 units	Gradual onset of polyuria and polydipsia, with development of mild acidosis
6	27	Father, maternal aunt	5 ft., 8 in.	205	?	?	C150-P80-F60; PZI 10 units.	Routine urine examination revealed sugar on admission to overseas hospital for circumcision
7	38	Mother	5 ft., 9½ in.	225	May, 1942	Oct., 1944	C110-P75-F40	Routine urine examination revealed sugar during hospitalization for gall-bladder disturbance prior to going overseas.
8	44	0	5 ft., 10 in.	200	Oct., 1943	Mar., 1944	C200-P90-F100	Routine urine examination revealed sugar
9	21	Paternal uncle	5 ft. 6½ in.	166	Mar., 1944	Mar., 1944	C150-P90-F80; PZI 26 units	Polyuria, polydipsia and acidosis developed after several weeks of combat duty, including continuous exposure to artillery fire
10	19	Maternal grandmother	6 ft., 1½ in.	170	Sept., 1944	Oct., 1944	C250-P85-F110; PZI 35 units.	Gradual onset of polyuria and polydipsia, and loss of 20 to 30 pounds in weight while overseas
11	43	0	5 ft. 6 in.	223	?	Sept., 1944	Regular diet	Based on glucose-tolerance test; patient asymptomatic, but had cirrhosis of liver.
12	24	Maternal grandmother	5 ft., 7½ in.	190	Dec., 1944	Jan., 1945	C200-P80-F90; PZI 17 units.	Onset of symptoms after considerable stress and strain while in an infantry unit in active combat
13	21	0	5 ft., 9 in.	180	Dec., 1944	Jan., 1945	C250-P100-F100	Gradual onset of polyuria while overseas
14	35	Mother	5 ft., 8 in.	212	1941	Feb., 1945	C200-P80-F90	Disease existed prior to service; diabetes rediscovered three years later when sulphur dermatitis proved refractory to treatment and urine examination revealed glycosuria.
15	36	0	5 ft., 8½ in.	200	1942	Feb., 1945	C200-P75-F70; PZI 25 units.	Migraine headaches for years, followed by gradual onset of polyuria and loss of weight; glycosuria found on routine admission for migraine.
16	40	Brother	5 ft., 3¾ in.	232	1941	Jan., 1945	C130-P65-F40	Onset prior to service, when patient had a carbuncle of neck and was treated with diet and small doses of insulin; a recurrence of polyuria Jan., 1945, while overseas.
17	43	0	5 ft., 8½ in.	154	?	May, 1945	Regular diet	Routine urine examination revealed sugar while awaiting separation from service
18	30	0	5 ft., 11 in.	215	Apr., 1945	May, 1945	PZI 10 units, RI 3 units	Gradual onset of polyuria and polydipsia, after dissatisfaction with job on return from overseas
19	28	Sister (died in coma)	5 ft., 6 in.	147	Mar., 1945	Apr., 1945	C190-P65-F100; PZI 40 units; RI 15 units.	Polyuria and nocturia; hospitalized for influenza; given penicillin treatment for latent syphilis; 1 wk. later admitted with vomiting and glycosuria and acetonuria found.
20	22	Maternal grandmother	5 ft., 4¾ in.	150	Dec., 1944	Apr., 1945	C200-P80-F100	Polyuria and increased thirst while on duty overseas
21	43	0	5 ft., 8 in.	228	1938	Mar., 1945	Regular diet	Diagnosis made at Walter Reed Hospital in 1938
22	54	0	6 ft., 1½ in.	190	June, 1944	Jul., 1944	C200-P85-F100	Polydipsia, nocturia, loss of weight and ulcer of foot while overseas
23	37	0	5 ft., 9 in.	214	?	Nov., 1944	C150-P75-F75	Dermatitis following administration of benzyl benzoate for scabies; routine urine examination revealed glycosuria.
24	38	0	5 ft., 8⅝ in.	175	May, 1944	Apr., 1945	C200-P90-F100; PZI 30 units.	Polyuria and nocturia while overseas
25	49	0	5 ft., 6 in.	180	1943	June, 1945	C175-P80-F90	Some increase in thirst and slight polyuria after entry into the service; routine urine examination revealed glycosuria at a separation center.

TABLE 2. *Data in Patients with No Known Precursors to Diabetes.*

CASE NO.	AGE	RELATIVES WITH DIABETES	HEIGHT	NORMAL WEIGHT	DATE OF ONSET	DATE OF HOSPITALIZATION	REGIME ON DISCHARGE	HISTORY OF ONSET
	yr.			*lb.*				
26	25	0	6 ft.	188	Oct., 1944	Nov., 1944	C233–P110–F105; PZI 30 units, RI 10 units.	Gradual onset of polyuria, polydipsia, polyphagia and weight loss while overseas
27	37	0	6 ft., 1 in.	188	Dec., 1944	Jan., 1945	C235–P90–F130; PZI 25 units, RI 8 units.	Gradual onset of polyuria and polydipsia, with loss of weight of 20 pounds, in Zone of Interior
28	22	0	5 ft., 8½ in.	162	Oct., 1944	Oct., 1944	C200–P80–F110; PZI 20 units.	Gradual onset of polydipsia and dryness of mouth 3 wk. after being withdrawn from combat
29	36	0	6 ft.	182	July, 1944	Aug. 7, 1944	C200–P90–F90; PZI 18 units.	Patient had severe emotional shock when buzz bomb struck company area; he became nervous and tired and began to lose weight in the next 10 days and developed polyuria and polydipsia.
30	21	0	5 ft., 8 in.	158	Sept., 1944	?	C200–P70–F80; PZI 20 units.	Patient was wounded in action polyuria and polydipsia developed during subsequent hospitalization.
31	29	0	6 ft.	170	Dec., 1944	Dec., 1944	C250–P100–F100; PZI 5 units.	When patient was inducted, he was told he had nervous tremors; he developed frequency on voyage overseas; immediately placed in active combat, when symptoms increased, and the patient lost weight, vomited and developed acidosis.
32	34	0	5 ft., 10 in.	165	Dec., 1944	Feb., 1945	C200–P90–F110; PZI 40 units.	Patient noticed fatigue, increased thirst, and nocturia while lost behind German lines for 5 days; these continued after he made his way to the American lines; he developed irritation of the penis a month later, glycosuria was found, and he was hospitalized.
33	26	0	5 ft., 7½ in.	?	Mar., 1944	?	C150–P90–F100; PZI 18 units.	Polyuria, polydipsia with gradual onset while overseas
34	34	0	5 ft., 10 in.	145	Jan., 1945	Mar., 1945	C225–P100–F90; PZI 14 units.	Patient wounded in Nov., 1944, and had two operations; nervous and jumpy while in Replacement Pool.
35	24	0	5 ft., 8 in.	150	?	Dec., 1944	C175–P90–F100; PZI 7 units	Routine urine examination revealed sugar on hospitalization for intercurrent illness
36	23	0	5 ft., 11 in.	160	Sept., 1944	Nov., 1944	C200–P90–F110; PZI 30 units.	Patient was under considerable mental and physical strain while trying to complete certain military duties prior to return to Zone of Interior on furlough
37	26	0	5 ft., 6½ in.	140	Oct., 1944	Nov., 1944	C200–P100–F100; PZI 40 units, RI 20 units.	Patient under considerable mental strain because of difficulties with commanding officer while overseas
38	24	0	5 ft., 8¼ in.	157	Apr., 1945	Apr., 1945	C200–P90–F90; PZI 15 units.	Polyuria, polydipsia and weakness developed while awaiting assignment in a parachute infantry unit
39	26	0	6 ft., 1¼ in.	185	Jan., 1945	Jan., 1945	C200–P90–F100	Gradual onset of polyuria, increased thirst and nocturia while working in a general hospital overseas
40	26	0	5 ft., 5 in.	140	Jan., 1945	Apr., 1945	C260–P76–F114	Patient developed polyuria and polydipsia while taking infantry basic training
41	24	0	5 ft., 8 in.	150	July, 1944	Apr., 1945	C200–P80–F120; PZI 20 units.	Gradual onset of increased thirst and loss of 25 pounds in weight while overseas
42	34	0	5 ft., 8 15/16 in.	168	Mar., 1945	May, 1945	C250–P70–F80; PZI 20 units.	Increased thirst and polyuria while waiting to cross Rhine with artillery outfit
43	21	0	5 ft., 6 in.	160	Feb., 1945	?	C200–P90–F100; PZI 30 units.	Patient developed "gas on stomach," polyuria, polydipsia and nocturia
44	24	0	5 ft.	104	July, 1944	July, 1944	Regular diet	Routine urine examination revealed sugar while overseas

1 case (Case 11), the diagnosis was doubtful. This patient had a definite compensated cirrhosis of the liver and a diabetic glucose-tolerance curve. We could not be certain that the disturbance in carbohydrate metabolism was not solely related to the underlying hepatic disease, but despite this fact the case was included in the series.

It was decided to classify the patients in two groups, comprising those with known precursors to diabetes and those with no known precursors. Inclusion in the former group was based on the factors of a family history of diabetes, of overweight and of age. The family history, which was gone into in great detail, was considered positive when a history of diabetes in the blood relatives, living or dead, was ascertained. An arbitrary 10 per cent increase in weight over that given in the height and weight table of the Association of Life Insurance Directors and Actuarial Society of America[14] was allowed. It was decided to include all patients over forty years of age in this group, because it was thought that these patients had reached the age when the incidence of diabetes increases. All but 2 of these patients were overweight.

The average age of the patients was thirty years, and the ages ranged from nineteen to fifty-four. Forty-three patients were men, and 1 was a twenty-four-year-old nurse. All the patients were white. Two were Jews; in neither of these did the diabetes begin after entry into the Service. In both cases, the diagnosis of diabetes had been made in civilian life, but the patients had been inducted into the Army. They were asymptomatic for two and a half and three years, respectively, at the end of which times the diabetes was rediscovered.

Two possible mechanisms suggest themselves by which psychogenic influences become capable of initiating diabetes. One of these is a functional neuroendocrine disturbance that gradually results in the development of diabetes. One would naturally expect that if this disturbance could be removed, the diabetic state would disappear or greatly improve, but no definite evidence of this sequence was seen. In many cases the diabetic state seemed to improve, but there was no certainty that this was not related to the better regulation of the diet that was possible in the Zone of Interior. The other mechanism is analogous to a trigger mechanism whereby the psychogenic factors initiate the diabetes and the condition, once it has begun, becomes permanent and irrevocable.

Menninger[12] sets down five requirements to which psychogenically induced diabetes should conform. These are an obvious psychopathic state evidenced prior to the development of the diabetic state; a mental picture quite different from the toxic state occasionally seen in hyperglycemia or hypoglycemia; a course of improvement in the mental picture paralleled by the glycemic and glycosuria levels, with fluctuations of these as emotional upsets occur in the psychic life; indication of the metabolic disorder by a persistent glycosuria, — without dietary control or insulin, — retarded glucose-utilization curves of the blood sugar and a response to dietary and in some cases insulin therapy; and clearing of the diabetes on mental recovery, without the necessity of insulin or rigid dietary treatment.

Complete psychiatric appraisals were not possible in these patients, but they assuredly suffered no marked psychiatric disturbance during their stay at this hospital. Nevertheless, they continued to take insulin, although the amount could be decreased. This decrease could also be explained on the basis of better regulation of the diet. There was no convincing evidence that the diabetes improved on the patients' return to the security of this country, and consequently it is not believed that these cases conform to the criteria set down by Menninger.

In addition, although we are well aware of the dangers of attempting to draw conclusions concerning the incidence of diabetes based on our statistics, we believe that diabetes is no more frequent in the Army than it is in civilian life. This is significant, since a soldier's life involves every kind of psychogenic trauma, including anxiety, insecurity and fear of death, so that a consequent increase in the incidence of diabetes is to be expected. On the other hand, a study of the history prior to the onset of the condition impresses one with the fact that in some of these cases an upsetting incident preceded the development of diabetes. As listed in Table 1, this was noted in 2 patients (Cases 9 and 12). In both these patients there was a family history of diabetes. Among the patients in whom there were no known precursors (Table 2), some (Cases 29, 30, 31, 32 and 34) showed suggestive relations, whereas in others (Cases 36, 37 and 40), the relations were less clear. It is significant that none of these patients recovered a normal sugar metabolism after being released from the stress of combat and realizing that they would soon be separated from the military service by a medical discharge. Follow-up information obtained in 11 patients (Cases 5, 6, 8, 10, 14, 27, 28, 29, 30, 36 and 37) four to six months after their return to civilian life revealed no appreciable change in their diabetic status.

Summary

A study was made of psychogenic factors preceding the onset of diabetes mellitus in 44 patients who were admitted to an army general hospital.

In no case was it possible to prove a direct causal relation between the stress incident to military service and the subsequent development of permanent diabetes.

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MEDICAL PROGRESS

UROLOGY

Carcinoma of the Prostate

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THE beneficial effects of a reduction of the male sex hormone (androgen control) in patients with carcinoma of the prostate were strikingly demonstrated by Huggins and Hodges[1] over four years ago. Since that time, a great many reports of the effects of castration and estrogen therapy on the malignant prostate have appeared. Although there is still no uniform opinion on the best method of treating this disease, there is a tendency toward agreement on certain points.

A wave of enthusiasm for castration in all cases of prostatic cancer followed these earlier reports. This was tempered by a few skeptics. On the other hand, castration and estrogen therapy have obviously been more effective in their immediate beneficial results than has any other treatment previously employed. The use of external or interstitial radiation has greatly decreased, and the partial removal of obstructing tissue by perineal operation has been largely abandoned in the treatment of prostatic cancer. From the many reports published during the last few years on series of cases of prostatic carcinoma treated in various ways, the questions arise whether castration or estrogen therapy is the better procedure, how effective and how permanent these methods are, and when these measures should be instituted.

There is fairly general agreement that the radical operation of Young, consisting of total perineal removal of the carcinomatous gland, including the capsule, prostatic urethra, bladder neck and seminal vesicles, is the only method that offers any expectation of cure. Objections to this procedure have emanated from those who believe that lymphatic extension has already taken place in practically every case of prostatic cancer when first seen, and from those who point out that few patients present themselves early enough to make this operation practical. It is true that the cancer should not have spread beyond the prostatic capsule, or at the most should involve only the lower ends of the seminal vesicles, if the radical operation is to be undertaken. If the results of radical surgery are good, however, it would seem that the patients who are found suitable for it, no matter how few, should not be denied its advantages. From the Brady Urological Clinic at the Johns Hopkins Hospital, Colston[2] reported that 23 per cent of the patients admitted to that institution with carcinoma of the prostate were subjected to radical operation. Probably less than 5 per cent of similar patients seen in most other clinics are found suitable for this operation. Previous reports from the same source have emphasized the value of radical surgery in early cancer of the prostate and bear sufficient evidence of long-term apparent cure to justify its use. According to Nathanson,[3] the following percentages of five-year cures of early cases of prostatic cancer treated by radical perineal prostatectomy have been reported: Belt, 58 per cent; Young, 53 per cent; and Colston, 50 per cent. Such figures appear to refute those who hold an entirely pessimistic attitude toward any possibility of cure of prostatic malignant disease.

It is also generally agreed that the majority of patients with cancer of the prostate are immediately benefited by castration or estrogen therapy. Bumpus et al.[4] recently reported 25 cases treated by orchidectomy, with improvement in all but 1. Huggins et al.[5] treated 21 cases by orchidectomy, with improvement in 15. Of 21 cases similarly treated by Marquart and Flaherty,[6] 11 were improved. Nesbit and Cummings[7] treated 75 patients

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by orchidectomy alone. Although 65 were improved clinically, 10 received no benefit whatever. Hamm[8] reported 5 cases all of which were helped by orchidectomy alone. These figures give an immediate improvement in 80 per cent of patients treated by orchidectomy alone.

There are those who favor estrogen therapy rather than orchidectomy. Kearns[9] treated 37 patients with stilbestrol, with improvement in 30. Kahle et al.[10] reported 7 patients benefited by stilbestrol alone, and of 12 patients so treated by Marquart and Flaherty,[6] 5 were improved. A case of spinal-fluid block relieved by stilbestrol alone was reported by Clarke and Viets.[11] Since approximately 75 per cent of patients treated with estrogens alone were improved, there appears to be little to choose between castration and estrogen therapy. Most writers believe, however, that the most spectacular and quickest beneficial results are obtained by orchidectomy. The issue is further confused by the fact that many authors have employed both castration and estrogenic therapy together.[12] It is obvious that more time must elapse before it becomes clear which form of treatment is the better.

Any hope that cancer of the prostate might be cured by endocrine therapy was dissipated long ago. All observers agree that the effects of such treatment are temporary and that sooner or later androgens from some source in the body again stimulate the cancer cells to activity. Regression of the primary growth and of metastatic lesions may be prompt and striking, and an almost unbelievable change for the better in the patient's physical condition may take place, but after months or a few years symptoms can be expected to reappear and the growth to become active again. It has been thought that the lesions that more closely resemble the adult gland respond best to treatment,[13] but others have found no correlation between the grade of malignancy and the length of relief afforded by treatment, and believe that lesions of higher grades of malignancy respond as well as, if not better than, those of lower grades.[14]

The expectation of life for those with cancer of the prostate, before orchidectomy or estrogen therapy was employed, was estimated by Bumpus[15] in 1926 from a study of 1000 cases at the Mayo Clinic. The average survival period was thirty-one months after diagnosis. Two thirds of those with metastases were dead nine months after their first treatment, and 58 per cent of those without metastases were dead in one year. In 1937, Welch and Nathanson[16] studied 204 cases of cancer of the prostate for life expectancy and reported that of 39 untreated patients, 25 per cent were dead in eight months, 50 per cent in twelve months and 75 per cent in twenty-one months after the onset of symptoms. Of the 175 treated patients, 25 per cent were dead in fifteen months, 50 per cent in twenty-six months and 75 per cent in forty-one months. They state that the British Ministry of Health found the average duration of life in 30 untreated cancers of the prostate to be less than seventeen months after the onset of symptoms.

The improvement in life expectancy that has resulted from the newer methods of treatment is shown by the following figures. Seventy-five patients with prostatic carcinoma treated by orchidectomy and carefully followed were reported by Nesbit and Cummings[7] in 1944. One third had died and two thirds were alive twenty-one months after operation. Eighty-four per cent of the patients with metastases passed the nine-month period in which Bumpus[15] reported a survival rate of only 33 per cent. Eighty-seven per cent of the patients without metastases were alive after twenty-one months, as contrasted with a survival rate of only 42 per cent after one year in Bumpus's series. When compared with Welch and Nathanson's figures of 50 per cent of treated patients alive after twenty-six months, the 66 per cent of total patients treated by Nesbit and Cummings by orchidectomy and alive after twenty-one months is less striking. The average duration of life of 21 patients with metastases who died after castration was only 11.3 months, although many of them were doubtless in an advanced stage of their disease. From the Mayo Clinic, Emmett and Greene[14] reported that 66 per cent of 133 patients were living eighteen months after orchidectomy and that 78 per cent of those with metastases survived the nine-month period. This again shows an improvement over Bumpus's figures, but the fact that 44 per cent of the patients were dead within eighteen months is not so impressive when compared with the 50 per cent of treated patients dead in twenty-six months as reported by Welch and Nathanson before androgen control. There seems little doubt that endocrine therapy prolongs the lives of patients with cancer of the prostate, but it is not yet clear just how much can be expected in this direction, particularly when the natural course of the disease varies so greatly.

The question of when endocrine therapy should be instituted is still being debated. A questionnaire was sent to seventy urologists in an effort to settle this point.[17] The answers were revealing in showing the confusion that exists concerning when treatment should be begun. One third of the urologists questioned perform bilateral orchidectomy as soon as the diagnosis of cancer of the prostate is made. Most of the rest delay it until metastasis can be recognized by x-ray, symptoms or laboratory evidence. A few do not favor the operation. From their observations, Nesbit and Cummings[7] conclude that the maximum benefit to the patient is derived by delaying endocrine treatment until the onset of symptoms from advanced or metastatic lesions has occurred. Others[13, 18, 19] agree with this.

There appears to be no definite evidence that castration or estrogen therapy prevents metastases.

In Nesbit and Cummings's[7] series, 31 per cent developed metastases after castration, in Emmett and Greene's series[14] 35 per cent did so. On the merits of combined treatment, orchidectomy and stilbestrol, Herger and Sauer[19] recently expressed the opinion that orchidectomy preceded or followed by stilbestrol does not offer any advantages over castration alone. Stilbestrol when given to patients whose symptoms recurred after orchidectomy was seldom helpful. They observed temporary benefit in 15 patients who had been on stilbestrol for a long time and then were castrated, whereas 4 showed no response. They conclude that castration after estrogen therapy may be of value, but that estrogen therapy begun after castration has failed is seldom effective.

Decapsulation of the Kidney

Opinions have varied considerably whether decapsulation of the kidney is beneficial in the treatment of certain renal conditions. The operation was advocated by Edebohls about forty years ago for chronic nephritis and was practiced for a while, but it soon lost its vogue because the results were disappointing. The benefits of renal decapsulation have been attributed to the production of a collateral circulation from the perirenal tissues, an increased blood supply from interruption of the sympathetic nerves and release of tension within the kidney. Whether such changes actually occur has been questioned. More recently decapsulation has been advised for renal lesions other than chronic nephritis, notably toxic changes due to bichloride of mercury poisoning, reactions from the sulfonamides and post-transfusion anuria. After a thorough review of this subject, Abeshouse[20] concludes that decapsulation has unquestionable merit in certain conditions and that the chief indications for its use are progressive oliguria and anuria. He advocates the operation in the nephrotic stage of chronic parenchymatous nephritis associated with uncontrollable edema and albuminuria, anuria from bichloride poisoning not relieved by other measures, anuria following sulfonamide therapy and post-transfusion anuria. Others who have had experience with these conditions, however, are not in agreement concerning decapsulation.

Deming[21] points out that although there is little difference of opinion concerning the chief indications for decapsulation, — anuria and oliguria, — there is no definite proof that the operation is the factor that increases the urinary output and that many patients recover who are not operated on. He is not at all convinced that patients with bichloride of mercury poisoning recover because of decapsulation. Several years ago, Talbott[22] expressed the following opinion concerning decapsulation of the kidney:

> The subject of decapsulation is of interest in a general discussion of nephrotoxic agents, since this form of therapy has been recommended in the treatment of anuria. It is my opinion that nothing is to be gained by decapsulating anuric kidneys. If resumption of urine flow follows decapsulation, restoration would probably have followed conservative treatment. Since decapsulation is an extensive operation and patients with anuria are acutely ill, additional insults may be fatal and are best avoided. Recently we had an unusual opportunity to study the effects of unilateral decapsulation in a young woman who developed anuria following a transfusion reaction. On the fourth day of anuria, the right kidney was decapsulated, and catheters were placed in both ureters. Resumption of urine flow began about twenty-four hours after operation. There was no difference, however, between the urine volume from the decapsulated kidney and that from the untouched organ. Six weeks later, after normal urine volume had been established for some time and the concentration of nonprotein nitrogen in the blood was almost normal, inulin and Diodrast clearances were done. The ureters were catheterized again so that each kidney could be studied separately. A marked reduction in the rate of formation of glomerular filtrate and in renal blood flow was observed. More significant was the comparison of the two kidneys. No striking difference between the kidneys was noted in any of the functions measured. A mathematical average of all functions, however, showed a slightly better performance by the untouched kidney in comparison with the decapsulated one. Our results are interpreted as implying that unilateral decapsulation in this patient had no beneficial action on renal function.

I know of no other observation on the effect of renal decapsulation so carefully controlled as this one. Since the patient mentioned above was operated on in 1942, I know of no other case of decapsulation for anuria at the Massachusetts General Hospital. There are, however, many who regard decapsulation as beneficial in the treatment of certain types of oliguria and anuria.

Hypertension and Nephroptosis

Of 133 cases of nephroptosis reported by Braasch and Goyanna,[23] only 16 showed hypertension, and in none of these was the nephroptosis considered the etiologic factor. The majority of patients with nephroptosis had blood pressures that were average or below average. Any compression of the blood vessels of the renal pedicle that might arise from the ptotic kidney was considered to be unlike the changes described by Goldblatt.

Endometriosis of the Bladder and Ureter

Invasion of the bladder by endometrial tissue has been considered unusual, and only 58 authentic cases have been reported in the literature. Twelve additional cases reported by O'Conor and Greenhill[24] give one the impression that this condition is not so rare as was supposed. Cyclic bleeding, considered a typical symptom, was recorded in only two thirds of the patients, and gross bleeding occurred much less frequently than one would expect. The cystoscopic appearance of these tumors may or may not be that of so-called "chocolate cysts." They are described as elevated, congested and edematous, often with a bluish or blue-black cast. The tumor mass may increase during menstruation. Biopsy is an aid in their detection. Removal of the ovaries usually results in a disappearance of these lesions, but occasionally this step does not

arrest the disease. In younger women in whom it is desirable to preserve ovarian tissue, localized excision of involved areas is advised. In women near the menopause, roentgen-ray irradiation of the ovaries usually causes the bladder lesions to disappear. If the lesions persist or progress after the removal of all ovarian tissue, segmental resection of the bladder wall is necessary.

Renal Calculi

Three years ago, a new technic for the operative removal of small stones in the kidney was described by Dees.[25] Since the recurrence of stones after pyelolithotomy is frequent and is due in many cases to failure to remove all small calculi at the time of operation, a method that helps to clear the kidney completely is of considerable importance. Dees's contribution may be regarded as a significant advance in the problem of renal calculi.

Following a prolonged, traumatic and apparently futile exploration of a kidney for a small elusive stone, as described by Dees, a blood clot was removed from the renal pelvis, within which the small calculus was entrapped. Impressed by this fortunate accident, he searched for a suitable coagulum to fill the renal pelvis and calices. Fibrinogen from human plasma fulfilled the requirements of a substance that would completely fill the interior of the kidney, clot quickly and firmly, not be harmful to the kidney and dissolve spontaneously. Fibrinogen solution is converted into a fibrin clot by adding thrombin. The thrombin used is a fraction of globulin derived from rabbit plasma. The coagulum formed is said to dissolve spontaneously in human urine at 37°C. within eighteen to twenty-four hours. At operation the upper ureter and renal pelvis are exposed. The ureter is gently occluded and a small incision is made in the renal pelvis. A No. 12 French urethral catheter is put into the pelvis, and any urine present is aspirated. The pelvis is washed with normal salt solution and then with fibrinogen solution. The pelvis is filled with fibrinogen through the catheter while simultaneously one tenth as much of 2 per cent clotting globulin is injected through the wall of the catheter with a syringe and needle. After five minutes, a firm cast of the pelvis and calices forms. This is removed by enlarging the pyelotomy incision. The coagulum frequently extrudes itself or may be removed by forceps, and brings with it all small stones that lie free within the kidney. Animal experiments conducted by Dees showed that no harm was done to the kidney by the retained coagulum or its introduction, and the procedure was carried out in 21 patients without demonstrable ill effect.[26]

We recently used the coagulum technic in a patient with multiple small stones in the kidney pelvis. The clot extruded itself intact on opening the pelvic wall, forming a perfect cast of pelvis and calices. All the calculi, including several small stones unsuspected in the preoperative x-ray films, were enmeshed in the clot. Air pyelograms assist greatly in determining beforehand whether this method is suitable, since unless the air surrounds all the stones in the calices of the kidney, the calculi are apt to be adherent and are not removed by the clot. Although this procedure has limitations, it has considerable value, and trauma to the kidney is minimal.

262 Beacon Street

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 32171

PRESENTATION OF CASE

A twenty-year-old unmarried woman entered the hospital because of progressive abdominal discomfort.

Three months before admission the patient noted a tendency to belch, especially soon after the evening meal and through the night. The eructations increased in frequency, and a month later the belched gas became foul smelling. Six weeks before admission nausea and excessive salivation developed, especially in the early morning. At about the same time the patient began to have intermittent diarrhea. Between periods of diarrhea the stools were usually normal, but on three occasions foul, black, sticky feces were passed. There were also intermittent episodes of abdominal discomfort and generalized crampy pain. She had lost 9 pounds in the two months before admission, although the appetite had been good. Two weeks before admission spontaneous vomiting immediately after meals began and persisted thereafter, although some meals were retained.

The last menstrual period began two months before admission and was normal; previously they had always been regular. The possibility of pregnancy was denied.

On physical examination there was diffuse, slight, lower abdominal tenderness accentuated in the subxiphoid region. There was a small, slightly tender, freely movable mass in the right lower quadrant near the midline. The breasts were slightly nodular.

The temperature, pulse, respirations and blood pressure were normal.

The red-cell count was 4,400,000, and the hemoglobin 12.7 gm. per 100 cc. The urine had a specific gravity of 1.028 and was normal. A guaiac test on the stool was negative. The gastric contents showed 12 units of total acid and no free acid before histamine and 11 units of total acid and 10 units of free acid after histamine. X-ray examination of the stomach was somewhat unsatisfactory because the patient vomited most of the barium; but a definite annular defect about 3 cm. long was seen in the antrum. There was only a very narrow channel through the antrum and pylorus leading to an essentially normal duodenal bulb. The rugal folds were swollen. The film of the chest was normal. Gastroscopy failed to visualize any lesion; peristalsis was normal, and there was no evidence of rigidity of the stomach wall within the range of the gastroscope.

On the third hospital day her family physician reported that an Aschheim–Zondek test done the day before entry was positive. Her symptoms continued unchanged.

An operation was performed on the eighth hospital day.

DIFFERENTIAL DIAGNOSIS

DR. MILTON H. CLIFFORD: From the story alone,—the skipping of a menstrual period and later a positive Aschheim–Zondek test, — one might have been justified in attributing the original symptoms to nervous indigestion precipitated by the emotional trauma and the subsequent symptoms of nausea and vomiting to the onset of pregnancy. Certainly the story is that of an increasing partial obstruction in the upper intestinal tract, presumably a pylorospasm from organic or functional causes, with periods of intermittent diarrhea suggesting irritation in either the lower intestine or in the large bowel. She passed foul, black, sticky feces. In the absence of taking iron or bismuth by mouth, the assumption is that the black feces represented bleeding and that she had had at least one episode of intestinal bleeding. This could not have been a constant oozing in view of the negative guaiac test noted later and in view of the level of hemoglobin, which, although not normal, was essentially so. There were two definite findings — x-ray evidence of a lesion in the stomach and a presumable lesion low in the abdomen on physical examination. Before we go farther, I should like to see the x-ray films.

DR. JAMES R. LINGLEY: This is a definite prepyloric lesion, which extends up both curvatures and involves about 3 or 4 cm. of the antrum. It is annular in type, and I think that the mucosa throughout this area is completely destroyed. There is also a suggestion of shelf formation along its upper margin.

DR. CLIFFORD: It is of interest that gastroscopy showed no abnormality, but of course the lesion was in the antral area, which is visualized with difficulty gastroscopically. The most significant negative information from the gastroscopic examination is that peristalsis was normal and that there was no sign of rigidity.

The second lesion that presented on physical examination, the small, slightly tender, freely movable mass in the right lower quadrant, near the midline, is consistent with a mass in the small bowel. It might, however, have been informative to have checked the colon by a barium enema. I am going to take that finding at its face value and assume

that it was probably present and that there were two lesions of the gastrointestinal tract that need to be explained.

For the moment let us say that she was pregnant and that her denial of pregnancy was false.

Dr. Tracy B. Mallory: It is only fair to say that, when presented with the results of the Aschheim–Zondek test, she changed her story.

Dr. Clifford: That also makes me change my story.

With a lesion in that area of the stomach and another lesion elsewhere in the bowel, the first thing that comes to mind is Hodgkin's disease or lymphoma. A lymphoma can cause intermittent profuse bleeding, and it can involve as great an area of the stomach without extreme symptoms as was demonstrated in this case by x-ray examination. As I thought this case over, before I knew that she was pregnant, the positive Aschheim–Zondek test made me entertain one wild speculation, which I shall still mention, to explain multiple intestinal lesions without pregnancy. The Aschheim–Zondek test is positive without pregnancy with a chorionepithelioma or hydatid mole. These usually occur following a pregnancy, but there have been some rare cases of chorionepithelioma of extragenital origin, which presumably arose from teratomas, with metastases in the lungs, liver and brain and some in the intestine. The possibility of implants of a chorionepithelioma in the intestines is ruled out by her admission of probable pregnancy, as well as by the rarity of such a pathological finding. A Krukenberg tumor or colloid carcinoma, with implants in the lower intestine, should also be mentioned in passing. An interesting feature is that she had not had more signs of intestinal bleeding; this presupposes that the lesion was primarily in the wall and was invasive, perhaps a sarcoma. We have no proof that the mass noted on physical examination was a second tumor, which thereby forces me to depend on the x-ray evidence. I shall make a provisional diagnosis of a sarcomatous lesion in the antrum.

A Physician: Was a pelvic examination done on this woman?

Dr. Lamar Soutter: Yes; she had an enlarged uterus, which was perfectly symmetrical and soft.

A Physician: Was there any connection between this and the abdominal mass?

Dr. Soutter: No; the abdominal mass was much higher than the record leads one to believe.

A Physician: Why does Dr. Clifford say sarcoma instead of carcinoma?

Dr. Clifford: Primarily because of the extensive annular growth, the three-month story and the patient's age.

Dr. Mallory: How would the appearance of this lesion in the stomach differ from that of an ectopic pancreas in the antrum, Dr. Lingley?

Dr. Lingley: Ectopic pancreatic tissue may produce a filling defect in the stomach that simulates carcinoma. The defect, however, is usually not annular but occurs rather more on one side.

Dr. Mallory: Sometimes it produces an annular swelling of the sphincter, almost like hypertrophic pyloric stenosis.

Dr. Lingley: In this case there was ulceration of the mucosa, so that I have no hesitation in calling it a malignant lesion.

Dr. Soutter: Preoperatively we were quite uncertain what this patient had, but we thought that benign hypertrophy was a good bet. We made a large incision for exploratory purposes, and when we got in, we found a large mass at the pylorus. The peritoneum over it was glistening and intact. There were quite a number of enlarged nodes along the lesser curvature. The pylorus was obviously enlarged with growth of some sort, but whether or not it was pancreatic tissue we did not know. There was no muscular hypertrophy. We sent for a pathologist and gave him two nodes from along the lesser curvature. He could not make a diagnosis but thought that the lesion was probably benign. We believed that she should have a resection of the stomach because of obstruction. As there was a possibility that this was a malignant disease, it seemed that, to be adequate, the resection should be high. Since such a resection in that age group would not increase the mortality over that of a low resection, we did a high one. In order to get the anastomosis to lie transversely, with the best functional effect, we also had to remove a large amount of the greater curvature. We had difficulty at the lower end because the pancreatic tissue was close to the lesion, and we had to resect some of the pancreas with the specimen.

Clinical Diagnosis

Carcinoma of stomach?
Sarcoma of stomach?

Dr. Clifford's Diagnosis

Sarcoma of antrum of stomach.

Anatomical Diagnosis

Carcinoma of stomach with metastasis to one lymph node.

Pathological Discussion

Dr. Mallory: We were unable to make a diagnosis on the gross specimen of the resected stomach. It did not seem to be as indurated or firm as we ordinarily expect a carcinoma to be. The possibility of lymphoma entered our minds. When the sections came through, it was quite obvious that the tumor was a scirrhous colloid carcinoma, which showed extensive infiltration of the wall of the stomach, and a single node out of the seven examined showed a small metastasis.

DR. CLIFFORD: How much mucosal involvement was there?

DR. MALLORY: The mucosa was ulcerated away over the entire extent of the tumor.

Can you answer the question about the second mass, Dr. Soutter?

DR. SOUTTER: There was no second mass.

CASE 32172

PRESENTATION OF CASE

A fifty-five-year-old auto-supply dealer entered the hospital complaining of jaundice and abdominal swelling.

At some indefinite period five or six months before admission the patient began to be troubled by "gas" in the abdomen and by increasing weakness. He continued to work until ten weeks before admission, when these symptoms, along with occasional nausea and vomiting, forced him to bed. Five weeks later he began having paroxysms of coughing and developed dyspnea. Two weeks before admission he became aware of a progressive uncomfortable swelling of the abdomen and of yellowness of the skin. He had never noticed any unusual color of the stools or urine. He complained of shooting pains in the arms and fingers and noticed tingling of the tip of the tongue. There had been marked wasting of the face, extremities and thorax, and an overall weight loss of some 15 pounds.

For ten or fifteen years the patient had consumed a fifth of a gallon of whisky daily. During that time his appetite was poor and he frequently went for a week at a time without eating any appreciable amount of food. He stopped drinking two weeks before admission.

Three and a half years before admission the patient entered another hospital because of gastrointestinal hemorrhage manifested by the passage of tarry stools. He received a blood transfusion and was discharged after a week, an x-ray diagnosis of duodenal ulcer having been made. There was no known gastrointestinal hemorrhage after that except for intermittent and, at times, fairly well marked bleeding from hemorrhoids. At the age of twenty-one years, the patient had jaundice, weakness and anorexia, which caused him to stop work for two months.

On physical examination the skin and scleras were moderately jaundiced. The tongue was beefy red, and the finer papillae were atrophied. A few fine crackling rales were heard at the base of the right lung. The protuberant abdomen bulged at the flanks, and there were a fluid wave and shifting dullness. The liver edge descended six finger-breadths below the ribs. A few spider angiomas were present on the skin of the chest and neck. There was moderate pitting edema of the legs and over the sacrum. All the extremities were weak, and the outspread hands trembled. There were prominent internal and external hemorrhoids.

The temperature was 99.6°F., the pulse 125, and the respirations 20. The blood pressure was 120 systolic, 70 diastolic.

The red-cell count was 2,850,000, with 8.8 gm. of hemoglobin. The white-cell count was 33,700, with 88 per cent neutrophils. The urine showed a specific gravity of 1.016 and gave a + test for albumin and bile; the sediment contained 10 white cells and a rare red cell per high-power field. Urobilinogen was demonstrable in a dilution of 1:200. A stool gave a negative guaiac reaction. The serum bilirubin level was 3.4 mg. per 100 cc. direct and 4.8 mg. indirect. Other blood chemical findings were as follows: nonprotein nitrogen, 21 mg. per 100 cc.; protein, 4.2 gm., with 2.3 gm. of albumin and 1.9 gm. of globulin; and vitamin A, 0.3 unit per cubic centimeter. A cephalin flocculation test was equivocal at twenty-four hours and ++++ at forty-eight hours. The prothrombin time was 33 seconds (normal, 19 to 22 seconds).

On the second hospital day 700 cc. of clear yellow ascitic fluid was obtained. Anorexia and weakness persisted. On the sixth day the patient was found sitting up in bed breathing deeply. He was drowsy and confused, complaining of numbness in the hands and feet. The serum bilirubin had risen to 5.2 mg. per 100 cc. direct and 7.8 mg. indirect, and the carbon dioxide level was 20.6 milliequiv. per liter. The chloride was 98 milliequiv. per liter, and the sodium 136.1 milliequiv. With intensive dietary and intravenous therapy his condition improved slightly over the course of weeks. Anorexia and apathy made it impossible for a satisfactory high-calorie intake to be maintained. The liver edge was felt to be definitely receding. On the thirty-second hospital day the patient gradually became markedly dyspneic and cyanotic. The pulse was unobtainable, and the blood pressure was 88 systolic, 70 diastolic. He was sweating and apprehensive and complained bitterly of abdominal distress. Attempts to pass a Levine tube were abandoned because the patient could not swallow and became even more cyanotic during the procedure. Respirations slowed and eventually ceased about six hours after the onset of the severe dyspnea.

DIFFERENTIAL DIAGNOSIS

DR. CHARLES L. SHORT: With the possible exception of the episode of gastrointestinal bleeding that occurred three and a half years before this patient's admission, his entire medical history seems to point to disease of the liver. The story of a high alcoholic intake for ten or fifteen years and, perhaps more important, his periodically inadequate diet form a sufficient cause for the development of liver failure. The appearance of his tongue and the pains and weakness of his extremities are further examples of the development of a deficiency syn-

drome of which hepatic cirrhosis is probably the most important part. The physical findings of jaundice, an enlarged liver, ascites, edema and spider angiomas are consistent. The laboratory findings are also consistent, so far as the low serum albumin, the positive cephalin-flocculation test, the low vitamin A in the serum and the increased prothrombin time are concerned. With the data at hand there is no way of determining whether the anemia was due to the deficiency or to blood loss from hemorrhage. The blood chemical findings at the time of his episode of dyspnea and confusion on the sixth day after examination resemble those reported in experimental liver injury, and in such cases the acidosis is said to be due to failure of the liver to convert lactic acid to glycogen. The only unusual laboratory finding in this case is the high white-cell count, which I am unable to explain on the basis of infection, either in the liver or elsewhere, or as due to a recent acute hemorrhage. Although a low white-cell count is almost the rule in cases of what might be called chronic cirrhosis of the liver, I believe that with more acute episodes of liver-cell destruction leukocytosis may be present. I remember one case of portal cirrhosis in which there was an unexplained white-cell count of 18,000; when the patient came to autopsy no other cause for the increased count could be found.

Is it possible to explain this picture on any other basis than that of cirrhosis of the liver? The large size of the liver suggests a malignant tumor, presumably metastatic. We are not told the character of the liver edge, but I think that the manifestations of liver insufficiency are too marked to make this as a single diagnosis; in addition, there is no evidence of a primary focus. A palpable spleen would be helpful at this point as further evidence against the liver enlargement's being due to metastatic malignancy. The only possible type of new growth that would fit in here, I believe, is a primary hepatoma superimposed on cirrhosis, but there are no positive findings by which one could logically make this diagnosis. It does not seem necessary to consider obstructive jaundice or, except for the increased white-cell count, an infectious process in the liver.

If we accept the diagnosis of cirrhosis of the liver, the next question concerns its type. The episode of jaundice at the age of twenty-one, which persisted for two months, brings up the probability that this patient died of a toxic or nodular cirrhosis, with the primary insult to the liver occurring many years previously. The large size of the liver at the time of admission is somewhat against this diagnosis and in favor of the more frequent alcoholic cirrhosis, but we are often reminded in the e exercises of the fallibility of physical diagnosis when it comes to determination of the size of the liver. Toxic cirrhosis, which is also called subacute or healed yellow atrophy and usually represents the end results of repeated episodes of liver-cell damage or destruction, also explains the leukocytosis.

There are two final points. One is whether or not the patient had a duodenal ulcer either three and a half years before or at the time of death. I do not believe that this can be answered, since he did not have a gastrointestinal x-ray examination during his hospital admission, which, incidentally, might also have shown the presence of esophageal varices. The second point concerns the terminal episode that immediately preceded his death, and this seems to me to be a vivid example of massive gastrointestinal hemorrhage. This presumably took place from a ruptured esophageal varix, although again one wonders remotely about the duodenal ulcer. He did not vomit blood, but this I believe has been recorded not infrequently in patients dying from such a cause.

I propose a diagnosis of liver failure due to toxic cirrhosis, with death resulting from hemorrhage from a ruptured esophageal varix.

Dr. Tracy B. Mallory: Dr. Volwiler, would you care to comment about the impressions on the ward?

Dr. Wade Volwiler: The protein level of the blood, with the normal albumin-globulin ratio and the slightly lowered vitamin A level, did not help much with the diagnosis. They were just consistent with malnutrition.

We treated this man for acute liver disease, without success. He refused to eat, and regardless of whatever one gives in the way of supplements and fancy medicines, I am sure that the fundamental basis of liver-disease therapy comes down to a proper diet and enough of it.

I agree that an increased white-cell count can be explained by massive and diffuse hepatic-cell necrosis. In such patients white-cell counts up to 20,000 or 30,000 are not infrequent, with a preponderance of neutrophils, a shift to the left and many toxic granules.

Clinical Diagnosis

Alcoholic cirrhosis of liver.

Dr. Short's Diagnoses

Toxic cirrhosis of liver, with esophageal varices.
Terminal gastrointestinal hemorrhage.

Anatomical Diagnoses

Cirrhosis of liver, alcoholic type.
Hemorrhages in lungs.
Bronchopneumonia.
Peritonitis, acute generalized early.

Pathological Discussion

Dr. Mallory: The autopsy showed cirrhosis of the liver. The liver at that time, whatever it may have been earlier, was small, weighing 1450 gm., perhaps 300 gm. below what might have been ex-

pected in a man of his size. It showed a diffuse cirrhosis with an extremely fine type of granularity, the characteristic gross picture of the cirrhosis seen in people with severe alcoholic histories. That was further borne out microscopically. In the early stages of alcoholic cirrhosis, large livers with intense fatty infiltration are the rule, and in the later stages the fat frequently disappears; but in both stages a characteristic type of hyaline degeneration is found in the liver cells. This is present in the great majority of the alcoholic cirrhoses and is rarely seen in the livers of those who do not give a history of alcoholism or at least in whom the possibility of alcoholism can really be excluded. I think that one is justified without question in making a diagnosis of alcoholic cirrhosis here. In contrast to many cases that one sees in the late atrophic stage, the process was still extremely active. A great many of the liver cells were currently undergoing this peculiar form of degeneration, and others were frankly necrotic. There were a great many foci of leukocytes about the necrotic cells, which fits in well with the elevated white-cell count.

The other features of the case were relatively unimportant. The spleen was not so much enlarged as one would expect in view of the chronicity of the story; it weighed only 250 gm. The heart was slightly hypertrophied. There were widespread hemorrhages throughout the lungs, which were probably due to liver insufficiency. The kidneys were essentially normal. There was terminal infection — a frank pneumonia and an early peritonitis.

DR. SHORT: Did he have esophageal varices?

DR. MALLORY: Small varices were found but apparently none had ruptured, since there was no blood in the stomach or intestines.

The New England Journal of Medicine

Formerly

The Boston Medical and Surgical Journal

Established 1828

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Subscription Terms: $6.00 per year in advance, postage paid, for the United States (medical students, $3.50 per year); Canada, $7.04 per year (Boston funds); $8.52 per year for all foreign countries belonging to the Postal Union.

Material should be received not later than noon on Thursday two weeks before date of publication.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to the *New England Journal of Medicine*, 8 Fenway, Boston 15, Massachusetts.

THE INADEQUACIES OF MEDICAL CARE

III. Those Concerning Its Quality

One topic rarely mentioned by the medical profession in discussions concerning medical care is its quality. It seems to be assumed that, because the science of medicine has advanced as far in the United States as in any other country of the world, all licensed physicians are equally proficient. Such, unfortunately, is not the case, and many things must be accomplished before the matter can be corrected.

The first step is to be sure that all medical students receive an adequate education. Although it is true that the majority of substandard schools have been forced to close, and that all the states but one do not permit graduates of such schools to take examinations for licensure, the fact remains that many thousands of graduates of substandard schools are in active practice. Undoubtedly some of these physicians give better medical care than do certain graduates of approved schools, but the majority of them do not. There has been no practical way in which any kind of check could be made on their activities, since the majority are not acceptable as staff members of recognized hospitals, a matter that is discussed in greater detail below.

A corollary of adequate medical education is the responsibility of the medical school and teaching hospital to turn out physicians whose training has been in accordance with the existing demand. Undoubtedly the majority of medical-school graduates aspire to become certified by one of the specialty boards, — in fact, 80 per cent of the 21,000 medical officers serving with the armed forces so stated in a poll taken two years ago, — but it is obvious that such a superfluity of specialists would not serve to the best interest of caring for patients. The bulk of the profession must be composed — as it always has been — of physicians who are trained to give general medical care and who are sufficiently intelligent and self-effacing to refer their difficult cases to the specialist. This type of medical education requires only about half the time necessary for qualification as a diplomate, and if an appreciable number of medical schools and teaching hospitals were to foster and promote such training, great benefit would accrue.

Secondly, some attempt should be made to control the various cultists who are licensed to practice their particular vocations in many of the states. Nothing short of a change in laws can remedy this situation, but one shudders to think of how many cases of tuberculosis and cancer remain unrecognized, because of this, until it is too late for effective therapy. In spite of her tardiness in finally refusing to accept applications for licensure from graduates of substandard medical schools, it is to the credit of Massachusetts that, to date, her legislators have refused to recognize cultists, other than osteopaths, and even they are required to take the same examination as do the graduates of regular medical schools.

A third difficulty arises from the fact that mere graduation from an approved medical school is no guarantee of an efficient and up-to-date practitioner. The science of medicine has advanced and is advancing so rapidly that a graduate of five or ten years is neglecting the majority of his cases of serious illness if he applies to them the knowledge that was his on graduation. He must constantly keep himself professionally "fit" — by reading, by refresher courses and by contact with his associates. This, of course, is much easier for the physician on the staff of a large hospital, particularly a teaching unit, but it is still the responsibility of all who consider themselves to be practitioners of medicine. Unfortunately, as previously mentioned, graduates of substandard schools, the very men who would benefit most by contact with others, are usually denied hospital privileges. One hospital in Massachusetts has already taken steps to remedy this situation. All such physicians within the community have been invited to attend ward rounds, staff meetings and clinicopathological conferences. Those who give evidence of a fair amount of medical knowledge and of a desire to improve themselves will be permitted to take patients to the hospital and to care for them, under supervision, except for major surgery and operative obstetrics. As a result, these men should show marked professional improvement, their patients will be assured adequate hospital facilities and the general health of the community should be raised. On the other hand, there will undoubtedly be some men who will scorn this help or who will be unable to profit by it; such practitioners obviously contribute little toward improvement of medical care. The adoption of this constructive scheme in other community hospitals deserves serious consideration; it has received the enthusiastic support of the Massachusetts Medical Society and the Massachusetts Hospital Association.

Another problem tied up with the quality of medical care concerns the question, Who is qualified to do what? Obviously a surgeon in a small community ordinarily should not perform a Wertheim operation, nor should he remove a tumor of the brain or lung. But there is nothing to prevent him from doing so if his regard for the well-being of the patient is so slight that he refuses to refer the case to a properly qualified surgeon. The American College of Surgeons and the American Board of Surgery have done much in recent years to label those who have had adequate training in surgery, and even boards of surgical specialists have been established. But all this accomplishes little unless the layman is taught that what one surgeon is capable of doing is not necessarily within the ability of another.

The final inadequacy that lowers the quality of medical care is a lack of diagnostic facilities. If the modern physician is denied the help of x-ray, chemical, pathological and bacteriological laboratories, his ability to diagnose most of his cases of serious illness falls to a level that is comparable with that of the family doctor of fifty years ago, — if, indeed, it does not fall lower, — and treatment of the patient accordingly suffers. Such laboratories can be maintained only by groups of physicians, clinics and hospitals, and this is undoubtedly one of the chief reasons why recently graduated physicians shun small cities and rural communities. The banding together of physicians in groups and the establishment of well equipped health centers and hospitals in areas that need them would do much to relieve the situation.

Several of the factors contributing to the quality of medical care concern state boards of licensure and departments of health. On the other hand, two of them — keeping professionally "fit" and practicing according to one's ability — are the direct responsibility of the practicing physician. Undoubtedly, however, the health of the Nation would be improved if the layman could be taught how to obtain the medical care that he specifically needs.

QUARTER CENTURY OF PROGRESS

A LETTER appearing elsewhere in this issue of the *Journal* serves at least to call attention to one interesting fact, namely, that the year 1946 marks the twenty-fifth anniversary of the Massachusetts Medical Society's ownership of the *Journal*. It may not be amiss to state that they have been years of mutual progress.

One hundred and thirty-five years ago the need for some outlet for the literary, scientific and social productivity of the Society other than the simple publication of its transactions was appreciated. Thus, on June 5, 1811, a committee was appointed "to ascertain, if possible, who are the conductors of a publication entitled *The New England Journal of Medicine and Surgery*, with a view to determine the expediency of incorporating the Communications of the Society in the said publication." It is assumed that the conductors preserved their anonymity, for no further report was forthcoming.

History tells us, however, that *The New England Journal of Medicine and Surgery and the Collateral Branches of Science*, a quarterly, continuing after 1826 as *The New England Medical Review and Journal*, was united in 1828 with *The Boston Medical Intelligencer*, which had been published as a weekly journal since 1823, to form *The Boston Medical and Surgical Journal* — a name of hallowed memory and a journal, we trust, of continuing and increasing usefulness, published since 1928 as *The New England Journal of Medicine*. Thus the publication record of the *Journal* stretches back uninterruptedly to 1812, when *The New England Journal of Medicine and Surgery* made its first appearance — an endurance record for the New World!

The Society continued to publish its transactions independently until 1914, although in 1847 its president, Dr. Jacob Bigelow, proposed that when the permanent fund of the Society amounted to $10,000 the income should be devoted to the publication of a journal. This matter was indefinitely postponed, but on July 1, 1914, a direct affiliation with *The Boston Medical and Surgical Journal* went into effect, whereby a special department in that journal was created, under the editorship of the secretary of the Society.

The question of ownership of the *Journal* did not reach an encouraging phase until 1920, when the Council voted to enter into negotiations for its purchase. The current owners proving receptive, the *Journal* eventually passed into the hands of the Society for the sum of one dollar and other, no doubt less obvious, considerations. The reorganization took place in April, 1921, and Dr. Walter P. Bowers became the managing editor.

During the first year of the Society's proprietorship of the *Journal*, all members were assessed five dollars to defray part of the costs of publication. They thus received, whether or not as individuals they wanted it, a six-dollar journal for a dollar less than the regular subscription rate. This assessment has gone down as the circulation and advertising rates have gone up, until finally in 1945, as our correspondent states, not only did the members of the Society receive the *Journal* without cost, but the periodical actually made money for the Society. The circulation of the *Journal* has increased from some 4500 in 1921 to over 18,000 in 1946.

The editors accept the probability that the precise terms in which the Treasurer's report is necessarily couched are somewhat misleading, but they have ordinarily neither the time nor the inclination to be greatly disturbed over it.

MASSACHUSETTS MEDICAL SOCIETY

MARKING OF PRESCRIPTIONS FOR HABIT-FORMING DRUGS

The following statement from the Massachusetts Board of Registration in Pharmacy relative to the proper marking of prescriptions for habit-forming drugs should be of interest to all practicing physicians.

MICHAEL A. TIGHE, *Secretary*

* * *

The Massachusetts Board of Registration in Pharmacy wishes to inform you that the Pure Food and Drug Division of the Federal Security Agency has recently ruled that Sections 502(*d*) and 503(*b*) relative to the marking of prescriptions ("Warning — May be habit forming") must be carried out to the letter of the law.

For your information, these sections of the law read as follows:

Section 502. A drug or device shall be deemed to be misbranded . . . . (*d*) If it is for use by man and contains any quantity of the narcotic or hypnotic substance alpha eucaine, barbituric acid, beta-eucaine, bromal, cannabis, carbromal, chloral, coca, cocaine, codeine, heroin, marihuana, morphine, opium, paraldehyde, peyote, or sulphonmethane; or any chemical derivative of such substance, which derivative has been by the Administrator, after investigation, found to be, and by regulations designated as, habit forming; unless its label bears the name and quantity or proportion of such substance or derivative and in juxtaposition therewith the statement "Warning — May be habit forming."

Section 503. . . . (*b*) A drug dispensed on a written prescription signed by a physician, dentist or veterinarian (except a drug dispensed in the course of the conduct of a business of dispensing drugs pursuant to diagnosis by mail), shall if —

(1) such physician, dentist or veterinarian is licensed by law to administer such drug, and

(2) such drug bears a label containing the name and place of business of the dispenser, the serial number and date of such prescription, and the name of such physician, dentist or veterinarian, be exempt from the requirements of Section 502 (*b*) and (*e*) and (in case such prescription is marked by the writer thereof as not refillable or its refilling is prohibited by law) of Section 502 (*d*).

These two sections are self-explanatory, but it should be emphasized that it is necessary to place on the container the following, "Warning — May be habit forming." This inscription is required on every prescription item, including refills if the prescribing physician does not bar a refill. In other words, the only time that the pharmacist does not put the "Warning — May be habit forming" label on a prescription is when the doctor marks the prescription "Not to be repeated."

DEATHS

ELLIOTT — John J. Elliott, M.D., of West Roxbury, died March 29. He was in his fiftieth year.

Dr. Elliott received his degree from Hahnemann Medical College and Hospital of Philadelphia in 1921. He was a member of the New England Obstetrical and Gynecological Society and the American College of Surgeons.

His widow survives.

PIPER — Frank J. Piper, M.D., of Framingham, died April 4. He was in his forty-ninth year.

Dr. Piper received his degree from Tufts College Medical School in 1924. He was a veteran of the Southwest Pacific, having seen service as a flight surgeon at Guadalcanal, the Admiralties and the Philippines. At the time of his death Commander Piper was on terminal leave after serving nearly four years in the Navy Medical Corps. Before entering service he was president of the staff and assistant to the surgical service of the active staff of the Framingham Union Hospital.

His widow, three daughters, his parents and a sister survive.

ROSENAU — Milton J. Rosenau, M.D., of Chapel Hill, North Carolina, died April 9. He was in his seventy-eighth year.

Dr. Rosenau received his degree from the University of Pennsylvania School of Medicine, Philadelphia, in 1889. For twenty-five years he was connected with Harvard Medical School, first as professor of preventive medicine and hygiene and later as professor of epidemiology at the Harvard School of Public Health. From 1913 to 1922 he was director of the School of Public Health of Harvard University and the Massachusetts Institute of Technology. He was a world renowned authority on preventive medicine and sanitation, and since 1935 had been director of the University of North Carolina School of Public Health. He was a fellow of the American Medical Association and a member of the Association of American Physicians and the Society of American Bacteriologists.

His widow, a son, a daughter and a stepson survive.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

NOTES

John J. Poutas, M.D., M.P.H., has resumed his duties as director of the Division of Local Health Administration, Massachusetts Department of Public Health, having returned from overseas duty in the European Theater. Prior to returning to this country, Lieutenant Colonel Poutas served as chief of the Epidemiology Branch of the Preventive Medicine Division, Office of the Theater Chief Surgeon. His duties will include supervision of the eight district offices of the department and the nursing, nutrition, social-service and sanitation bureaus.

Roy F. Feemster, M.D., recently acting director of the Division of Local Health Administration, Massachusetts Department of Public Health, has returned to his former position as director of the Division of Communicable Disease.

Dwight J. Mulford, Ph.D., has been appointed laboratory chief of the Blood Processing Laboratory in the Division of Biologic Laboratories, Massachusetts Department of Public Health. This laboratory is especially designed for the fractionation of blood plasma into albumin, immune serum globulin, fibrin foam, fibrin film and other fractions of value in medicine, surgery and public health.

Otto C. Yens, M.D., has been appointed supervisor in charge of Crippled Children Services in the Massachusetts Department of Public Health. Prior to his appointment, Dr. Yens held the rank of lieutenant colonel in the Medical Service of the 117th Evacuation Hospital, United States Army.

Ruth Alida Thomas, M.A., C.P.H., has joined the staff of the Bureau of Health Information, Massachusetts Department of Public Health. Miss Thomas was formerly assistant professor in the Department of Hygiene and Bacteriology, Smith College. She also served as consultant in the Medical Intelligence Branch of the Preventive Medicine Division, Office of the Surgeon General, United States Army.

Alexander A. Robertson, formerly chief sanitary officer with the Newton Health Department, is now serving with the Massachusetts Department of Public Health as supervising health-district sanitary officer.

John B. Skinner, M.S., has been appointed director of the Division of Occupational Hygiene of the Massachusetts Department of Labor and Industries. Mr. Skinner has been an engineer with the department since 1941. He succeeds Mr. Manfred Bowditch, who retired from state service to assume the position of field director of the Saranac Laboratory, Saranac Lake, New York.

CORRESPONDENCE

THE *JOURNAL* PAYS ITS OWN WAY

To the Editor: Why should not the *Journal's* financial success be fully recognized? Despite the fact that the members of the Massachusetts Medical Society have always received their copies of the *Journal* at a figure below the usual subscription rate, even from the earliest years of the Society's ownership, the annual report of the Treasurer has consistently ignored this basic fact and presented the fiscal affairs of the *Journal* as if the publication were run at a loss, the deficit being made up by a benevolent grant in the form of an appropriation by the Society.

It appears to me high time that the members of the Society realize that they have never paid full rates for their subscriptions, that their per capita annual assessment (call it "appropriation" if you like) has steadily decreased under the administration of two able managing editors, Dr. Walter P. Bowers and Dr. Robert N. Nye, and that in 1945 not only did they receive their copies of the *Journal* without cost, but the *Journal* actually made money for the Society. Let us give credit where credit is due and stop speaking, writing and thinking of the *Journal* as if it were a drain on the Society's resources.

JOSEPH GARLAND, M.D.

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NOTICES

ANNOUNCEMENTS

Dr. F. H. Capodieci, having been released from military service, announces the opening of an office for the general practice of medicine at 247 Harvard Street, Dorchester.

Dr. Leo J. Cass, having returned from military service, is resuming the practice of internal medicine at 1101 Beacon Street, Brookline, and 20 Ridgefield Road, Winchester.

Dr. Lewis S. Pilcher announces his return to the practice of general surgery with offices at 43 Parker Street, Newton Centre, and 12 Bay State Road, Boston.

Dr. George C. Prather, having returned from military service, announces the reopening of his office for the practice of urology at 1180 Beacon Street, Brookline.

NEW ENGLAND ROENTGEN RAY SOCIETY

The next meeting of the New England Roentgen Ray Society will be held at the Harvard Club of Boston on Friday, May 17, at 5:30 p.m. Dr. A. C. Christie will present the Holmes Annual Lecture, his subject being "The First Fifty Years of Radiology: The elements that have contributed to its growth as a great medical specialty."

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

The monthly clinical conference and meeting of the staff of the New England Hospital for Women and Children will be held on Thursday, May 2, at 7:15 p.m. in the classroom of the Nurses' Residence. Miss Bernice W. Billings, R.N., will speak on the subject "Program of the Boston Tuberculosis Association." Dr. Elizabeth O'Neil will be chairman.

(*Notices continued on page xv*)